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HEAD INJURY

NEUROLOGIC AND PSYCHIATRIC ASPECTS

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The subject of head injury has long interested physicians. Hippocrates [1] (born 460 B. C.) noted grave symptoms and contralateral convulsions following injury to the head. He attributed these changes to fracture of the bone and apparently not to injury of the brain substance. Commentators during the following centuries added little to his descriptions. Celsus [2] (25 B. C. to 50 A. D.) mentioned cerebral hemorrhage without injury of the skull after trauma to the head. Lanfrancus [3] (died about 1306), in the thirteenth century, wrote about "the brain when it is shaken by concussion or by severe beating without fracture of the skull or injury to the skin." Berengarius de Capri,[4] in 1517, also admitted that concussion may at times occur without fracture of the skull. Ambroise Paré,[5] a great admirer of Hippocrates, in 1575 ascribed the symptoms of commotio cerebri to rupture of the diploic and intracranial blood vessels, the putrefying extravasated blood affecting the cerebral tissues. He described the famous case of extracerebral bleeding in Henry II, who died following a blow to the head during a tourney. This keen observer also noted that clinical changes may come on some time after the initial injury.

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Read at the Fifty-Ninth Annual Meeting of the American Neurological Association, Washington, D. C., May 9, 1933.

1. Hippocrates: Genuine Works. Translated from the Greek with a Preliminary Discourse and Annotations by Francis Adams, London, Sydenham Society, 1849.

2. Celsus, A. C., cited by Gurlt, E.: Geschichte der Chirurgie, Berlin, A. Hirschwald, 1898, vol. 1, p. 379.

3. Lanfrancus: Science of Cirurgie, London, K. Paul, Trench [and others], 1894; Cyrurgia parva, tractatus 2, caput 1.

4. Berengarius de Capri, cited by J. F. Malgaigne,[5a] p. 24.

5. (*a*) Paré, A.: Oeuvres complets, with an introduction by J. F. Malgaigne, Paris, J. B. Baillière, 1840. (*b*) Paget, Stephen: Ambroise Paré and His Times, New York, G. P. Putnam's Sons, 1897.

He accounted for these by inflammation and putrefaction. Rembert Dodoens,[6] in 1581, noted laceration of brain tissue without any skull injury after a blow on the head. Boirel,[7] in 1677, attempted to delineate cerebral concussion clinically as a slight shake-up of the head without cerebral or external injury.

Littré's [8] famous case, reported in 1705, did much to give definite form to the conception of concussion. A young criminal threw himself against a prison wall and died immediately. On examination, the brain was somewhat contracted and dense, but the skull was intact. This proved to the physicians of that day that serious head trauma can take place with intact cerebral substance. As a result of this observation, concussion was definitely isolated from other cerebral injuries. It was considered apart, as not due to changes in brain tissue. The clinical picture of commotio cerebri remained unclear and ill-defined.

Interest was aroused in the question of head injuries in the middle of the eighteenth century as a result of a prize offered by the Paris Academy of Medicine for the best explanation for the contrecoup phenomenon. A few years before this time, a communication by Petit,[9] later incorporated in his surgical textbook, had considerable influence in molding medical thought regarding brain injuries. He stated that commotio cerebri is due to the transmission of the vibrations of the skull to the brain substance. This transmission of the blow to the brain substance is more severe without skull fracture. He also discussed in his textbook, published posthumously about 1790, the question of intracranial bleeding as a result of head injury, and he indicated the necessity for an interval of lucidity before the diagnosis of such a hemorrhage could be made. The influence of his ideas is seen in Boyer's textbook on surgery published during the early part of the nineteenth century.

Desault and Bichat [10] soon challenged Petit's clinical conceptions and suggested that bleeding following head trauma may occur immediately after the blow and that it is indicated by the presence of hemiplegia and of other paralyses rather than by the interval of freedom from symptoms. This correction was accepted by Nélaton [11] and other

6. Dodoens, Rembert, cited by Gurlt, E.: Geschichte der Chirurgie, Berlin, A. Hirschwald, 1898, vol. 3, p. 10.

7. Thomas, L.: Antoine Boirel, Gaz. d. hôp. **53**:717, 1880.

8. Littré, M., in Histoire de l'Académie royale des sciences, Paris, 1705, p. 54.

9. Petit, J. L.: Traité des maladies chirurgicales, et des opérations qui leur conviennent, Paris, Méquignon l'Aineé, 1790, vol. 1, p. 80.

10. Desault, P. J.: The Surgical Works or Statement of the Doctrine and Practice of P. J. Desault, by Xavier Bichat, Philadelphia, T. Dobson, 1814, vol. 1, p. 22.

11. Nélaton, A.: Clinical Lectures on Surgery, from Notes Taken by W. F. Atlee, Philadelphia, J. B. Lippincott Company, 1855, p. 315.

clinicians of the day. Boyer,[12] in 1822, Sir Astley Cooper,[13] in 1837, and Dupuytren, in his lectures in 1839, developed rather clearly the picture of acute commotio cerebri and began to emphasize the syndrome of persistent sequelae. Boyer stated that in certain of these patients the effect of concussion may even be permanent. Dupuytren [14] described in detail the somatic and psychic consequences of such injuries.

In 1866, there appeared a memorable contribution which upset what had been learned up to that time. Erichsen [15] published a summary of his experiences with railway injuries, in which he emphasized the importance and gravity of spinal concussion. This condition had been previously mentioned by older clinicians, among them Ollivier [16] and Abercrombie,[17] and was considered to be due to a disorganization of the activity of the spinal cord as a result of blows to the vertebral column without gross injury to the spine. Many of Erichson's patients presented cerebral symptoms. His explanation of the existence of these symptoms was an ascending meningomyelitis, extending upward to involve the intracranial structures. He used the term "molecular disarrangement" for the first time. This conception had much significance in directing the physiologic thought of subsequent students of this subject. Though his studies were grossly incomplete and though some of his cases were undoubtedly due to syphilis of the central nervous system and to organic nervous diseases, as Bailey [18] and Page [19] have pointed out, his writings are nevertheless worthy of mention. Neurologic semeiology was hardly developed at that time, and Erichsen was not able to distinguish disease of the spinal cord from that of the brain.

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12. Boyer, A.: Traité des maladies chirurgicales, et des opérations qui leur conviennent, ed. 3, Paris, chez l'auteur, Migneret, 1822, vol. 5, p. 89.

13. Cooper, Astley P.: Lectures on the Principles and Practice of Surgery, with Additional Notes and Cases by Frederick Tyrrel, Philadelphia, Haswell, Barrington & Haswell, 1839, p. 130.

14. Dupuytren, G.: Leçons orales de clinique chirurgicale faites â l'Hôtel-dieu de Paris, ed. 2, Paris, Germer-Baillière, 1839, vol. 5, p. 253. Allamac, A.: Dupuytren et la neurasthénie post-traumatique, Thèse de Paris, 1920.

15. Erichsen, J. E.: On Concussion of the Spine, Nervous Shock and Other Obscure Injuries to the Nervous System, new edition, Baltimore, William Wood & Company, 1886.

16. Ollivier, C. P.: De la moëlle épinière et ses maladies, ed. 2, Paris, Crevot, 1827, vol. 1, p. 410.

17. Abercrombie, John: Pathological and Practical Researches on Diseases of the Brain and the Spinal Cord, Edinburgh, Waugh & Innes, 1828, p. 375.

18. Bailey, Pearce: Diseases of the Nervous System Resulting from Accident and Injury, New York, D. Appleton and Company, 1908.

19. Page, H. W.: Injuries of the Spine and the Spinal Cord and Without Mechanical Lesions and Nervous Shock in Their Surgical and Medico-Legal Aspects, London, J. & A. Churchill, 1883.

Obersteiner's [20] demonstration, in 1879, of changes in the ganglion cells in the spinal cord of a soldier with paraplegia due to a bullet wound in the spine, which did not injure the cord, was of considerable importance in lending greater weight to the clinical assertions of Erichsen. Obersteiner also utilized the conception of molecular disorganization to account for the seriousness of the clinical picture in the presence of minimal anatomic findings. Erb,[21] in his article in Ziemssen's encyclopedia, and Leyden,[22] in his textbook on diseases of the spinal cord, accepted the teachings of Erichsen and confirmed from their experience the underlying clinical and pathologic implications of such a hypothesis. However, opposition to these views was soon to come. Rigler,[23] in 1879, noted a marked increase in invalidism resulting from railway accidents following the passage of the compensation law in Prussia, in 1871. He emphasized the rôles of initial terror, protracted idleness, hysteria and desire for compensation. He doubted the soundness of the conception of spinal concussion. Hodges,[24] at a clinical society meeting in Boston in 1880, discussed the erroneous impression regarding the severity of these spinal lesions and insisted that there was much exaggeration in the evaluation of the injuries. He suggested vascular changes in the cord as causing the clinical pictures usually encountered after railway accidents. Moeli,[25] in 1881, reported four cases of alleged spinal injury from the Westphal clinic in the Charité. All the patients showed mental changes. The author noted, with little confidence, the similarity of the mental picture to that seen after head injuries, noting at the same time the pathogenic significance of fright. The contribution is rather unclear and timid.

Page, in 1883, launched a vigorous attack on the accepted notions regarding spinal concussion and the concomitant cerebral changes. He denied the existence of meningeal lesions and asserted that there existed no case to establish unequivocally the reality of spinal concussion. He alleged that many of the reported cases were due to intraspinal hemorrhage and to causes other than trauma, such as syphilis. Without

20. Obersteiner, H.: Ueber Erschütterung des Rückenmarkes, Med. Jahrb., 1879, p. 531.

21. Erb, W. H.: Diseases of the Spinal Cord and the Medulla Oblongata, in von Ziemssen, H.: Cyclopedia of the Practice of Medicine, New York, William Wood & Company, 1878, vol. 13, p. 347.

22. Leyden, E.: Klinik der Rückenmarkskrankheiten, Berlin, A. Hirschwald, 1875, vol. 2, p. 99.

23. Rigler, J.: Ueber die Folgen der Verletzungen auf Eisenbahnen insbesondere der Verletzungen des Rückenmarks, Berlin, G. Reimer, 1879.

24. Hodges: Concussion of the Spine—So Called, Boston M. & S. J. **102**:132 (Feb. 6) 1880.

25. Moeli, C.: Ueber psychische Störungen nach Eisenbahnunfällen, Berl. klin. Wchnschr. **18**:73, 1881.

hesitation and with considerable conviction he pointed out the importance in the etiology of the symptoms following railway injuries of the temptation to exaggerate, imposture, fright and other "purely psychical causes."

Dana,[26] in 1884, in the United States, objected to Erichsen's generalizations. Strümpell,[27] in 1888, published a paper which has been widely read and frequently quoted in expert opinions in cases of so-called neurosis following trauma. He analyzed the significance of the desire for compensation and the ever present tendency to exaggerate and capitalize the accident. He indicated that a distinction must be made between the sequelae of head injuries and those of trauma to other parts of the body. In 1896, he epitomized his attitude[28] by saying: "I cannot free myself from a certain feeling of uncertainty in these cases of head injury." This rather definite distinction for the first time between the results of head injury and those of body injury was an important step in clarifying the issues involved in this hitherto complicated and vague subject.

Oppenheim,[29] in 1889, published his famous monograph on traumatic neuroses. This term had not been in general use previously. Oppenheim classified as traumatic neuroses all neuropsychiatric sequelae of injury that could not well be included in hysteria, neurasthenia or definite organic syndromes. He preferred to see in these cases the operation of organic and psychic factors and offered as an explanation the possibility of impairment of the function of the central nervous system by the propagation to it of strong afferent stimuli. A critical analysis of the case histories in this historic contribution shows that Oppenheim included in this nosologic entity all sorts of conditions, unclassifiable at the time because of the limitations of the clinical neurology of his day. One recognizes probable cases of neurosyphilis and of other organic diseases of the nervous system (cases 5, 18 and 42). The contemporaries of Oppenheim accepted his suggestion. Many of them regarded this condition as a new disease. The conception facilitated their clinical work and had definite practical value.

Soon after the publication of Oppenheim's work, opposing voices were heard, objecting to the use of the term "traumatic neurosis.

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26. Dana, C. L.: Concussion of the Spine and Its Relation to Neurasthenia and Hysteria, M. Rec. **26**:617 (Dec. 6) 1884.

27. Strümpell, Adolph: Ueber die traumatischen Neurosen, Berlin, Gustav Fischer, 1888.

28. Strümpell, Adolph: Ueber die Untersuchung, Beurteilung und Behandlung von Unfallkranken, Munich, J. F. Lehmann, 1895.

29. Oppenheim, H.: Die traumatischen Neurosen nach den in der Nervenklinik der Charité in den 8 Jahren 1883-1891, ed. 2, Berlin, A. Hirschwald, 1892.

Schultz,[30] at the Tenth International Congress of Medicine in Berlin, in 1890, declared that there was no need for the perpetuation of this term; Jessen,[31] in 1896, and Knapp,[32] in 1897, similarly remonstrated. Although in 1896 Nonne [33] chose to keep the term, in 1915, after two decades of vast neurologic experience, he emphatically urged its abandonment.

In spite of these many and repeated cogent objections, for the past four decades clinicians have been clinging tenaciously to this confusing conception. It is worth noting that during the World War Oppenheim stated that he still believed in his ideas on this subject as originally expressed.

Charcot [34] and his students did not espouse the teachings of Oppenheim. They noted that the clinical phenomena following traumatism of all types resembled changes noted during hypnosis. They proposed the theory that these changes were due to hysteria and neurasthenia. It is interesting to read, for example, the report of a case included by Charcot in his "Leçons du mardi" for 1888 and 1889, which later clinicians would not have hesitated to include in the group of organic posttraumatic syndromes. It is surprising how unhesitatingly he classified this condition in his hybrid rubric "hysteroneurasthenia" although there were definite loss of consciousness, complete amnesia for the accident, no reliving of the original trauma, headache and dizziness. But Charcot spoke with the voice of authority.

Up to this time, except for Strümpell, authors made little distinction between injury to the head and that to other parts of the body. When cerebral symptoms were present they were considered as due to ascending meningomyelitis, to a centripetal effect of strong afferent stimuli on the central nervous system or to a psychologic reaction to the trauma situation. Strümpell, in 1888, noted the necessity of distinguishing between sequelae due to organic changes within the nervous system and those secondary to mental factors.

In 1892, Friedmann [35] wrote an important paper in which he separated from the large, ill-defined group of the so-called traumatic

30. Schultz, F.: Tr. Internat. Cong. Med., Berlin, 1890, quoted by Rosenthal.[60]

31. Jessen: Diskussion über Nervenunfallserkrankungen, Neurol. Centralbl. **15**:569 (April 22) 1896.

32. Knapp, P. C.: Traumatic Neurasthenia and Hysteria, Brain **20**:385, 1897.

33. Nonne, Max: Soll man wieder traumatische Neurose bei Kriegsverletztungen diagnostizieren? Schlussbemerkung Med. Klin. **11**:849 (Aug. 1) 1915.

34. Charcot, J. M.: Leçons du mardi, à la Salpêtrière. Policliniques, 1888-1889, Paris, E. Lecrosnier & Babé, 1889, vol. 2, p. 131.

35. Friedmann, M.: Ueber eine besondere schwere Form von Folgezuständen nach Gehirnerschütterung und über den vasomotorischen Symptomencomplex bei derselben im Allgemeinen, Arch. f. Psychiat. **23**:230, 1892.

neuroses a number of cases characterized by the presence of headache, dizziness, vasomotor instability and intolerance to alcohol. He insisted that this symptom complex deserved nosologic independence, and he called it the vasomotor symptom complex. He considered the changes as due to a disordered intracranial circulation. Whether the case that he reported was really one of cerebrovascular syphilis is of little moment. The contribution was exceedingly valuable in directing attention again to the necessity for separate consideration of cases of injury to the head and to the probability of an organic basis for some of the cases with a posttraumatic symptom complex. In 1910,[36] he reviewed the subject in the light of his subsequent experience and corroborated his original views. Vibert,[37] in 1893, independently expressed a similar point of view and urged the necessity for the separation of cases of head injury from the other traumatic cases. He believed that the sequelae of head injury are due to actual alterations in the intracranial contents. Koeppen,[38] in 1897, and Neel,[39] in 1915, noted the similarity of the mental changes in cases of head injury to those seen in other organic diseases of the nervous system (dementia paralytica). In addition, Neel emphasized the rarity of such findings after peripheral injuries and pointed out that these disturbances were noted in cases of head injury long before the passage of any compensation law.

The need for separate discussion and treatment of head injuries was becoming recognized, and in 1916, Horn [40] pleaded again for the recognition of the difference between the organic sequelae of injury to the nervous system and the psychologic response to the "trauma situation." He also delimited in a clear manner the syndrome of terror neurosis (*Shreckneurose*) as distinct from the organic postconcussion neurosis (*Kommotionsneurose*). The same plea was reiterated by Tromner [41] in 1910 and again in 1921. He suggested the term "encephalopathia traumatica" for the definitely organic consequences of

36. Friedmann, M.: Ueber die materielle Grundlage und die Prognose der Unfallneurose nach Gehirnerschütterung, Deutsche med. Wchnschr. **36**:698, 1910.

37. Vibert, Charles: La névrose traumatique; étude médicolégale sur les blessures produites par les accidents de chemin de fer et les traumatismes analogues, Paris, J.-B. Baillière & fils, 1893.

38. Koeppen, D.: Ueber Gehirnveränderungen nach Trauma, Neurol. Centralbl. **16**:965, 1897.

39. Neel, A. V.: Ueber traumatische Neurosen deren späteren Verlauf und ihr Verhältnis zur Entschädigungsfrage, Ztschr. f. d. ges. Neurol. u. Psychiat. **30**:379, 1915.

40. Horn, Paul: Ueber Symptomatologie und Prognose der cerebralen Kommotionsneurosen unter vergleichender Berücksichtigung der Kopfkontusionen der Schädeldach-und Basisbrüche, Ztschr. f. d. ges. Neurol. u. Psychiat. **34**:206, 1916.

41. Tromner, E.: Erinnerungen an die traumatische Hirnschwäche (Encephalopathia traumatica), Deutsche Ztschr. f. Nervenh. **68-69**:491, 1921.

injury to the head. It was becoming clearer to students of this subject that the clinical picture after head trauma must be looked on as the resultant of the interaction of organic and psychogenic factors. After Horn's work the pathoplastic rôle of experience and personality peculiarities in the development of particular reactions, even in the distinctly organic syndromes, received the attention of investigators (Jelliffe,[42] encephalitis; Ferenczi and Hollis,[43] dementia paralytica; Kretschmer,[44] head trauma). It was becoming evident that the clinician's approach to these problems must take cognizance of the constant operation in every case of psychogenic, constitutional and organic factors.

The experiences of the World War did much to influence the attitude of the medical profession toward the sequelae of injuries to the nervous system. The gross impression of many clinicians was that most of the so-called neurotic manifestations were distinctly functional. This point of view persisted after the close of the war, as we shall show. It is interesting to note the enthusiasm of an eminent organic neurologist like Nonne as an arch-defender of the psychogenic origin of most of the nervous sequelae of war injuries. He was influenced by the ready response of many of his patients to hypnosis.

One must, however, consider the limitations of this war material as scientific data and reserve conclusions drawn from such observations until this vast experience has been envisaged from a critical point of view:

1. Frequently no distinction is made between injury to the head and that to other parts of the body. The details of the actual accident were often not available under the conditions of war.

2. The opportunities for careful clinical study and analysis of soldiers behind the lines were necessarily limited. The differential diagnosis between concussion of the brain and collapse as a result of intense fright could not be accurately made at times. There is, therefore, no question in our minds that cases of terror neurosis were repeatedly being confounded with those of true commotio cerebri.

3. The element of terror and continued anxiety present so prominently in soldiers during active warfare is apparently absent in most cases of head trauma in civil life. Most of the patients struck by automobiles or severely injured during the course of their work do

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42. Jelliffe, S. E.: Postencephalitic Respiratory Disorders, Washington, D. C., Nervous and Mental Disease Publishing Company, 1927.

43. Ferenczi, S., and Hollis, S.: Psychoanalysis and the Psychic Disorder of General Paresis, Washington, D. C., Nervous and Mental Disease Publishing Company, 1925.

44. Kretschmer, E.: Ueber psychogene Wahnbildung bei traumatischer Hirnschwäche, Ztschr. f. d. ges. Neurol. u. Psychiat. **45**:272, 1919.

not recall any details of the injury, and recollections of such accidents play no significant rôle in their dreams or in their mental content. This differs considerably from the usual experiences of those suffering from war neurosis. There is thus absence of the important initial psychogenic factors.

4. Marinesco [45] and Mott [46] pointed out the importance of carbon monoxide poisoning in the pathogenesis of syndromes following the explosion of large shells or mines.

5. It has been noted by many students of head injuries that the wounds resulting from small projectiles traveling at terrific speed are not comparable to the effects of the usual blow on the head in civil life, which causes a diffuse, duller trauma to the skull as well as considerably more jarring of the intracranial contents (Fuchs [47]).

Clinicians have too readily cited the experiences during the war to substantiate theories of their own. One must evidently be cautious in comparing this experience with the problems in times of peace. There was considerable difference of opinion regarding the nature of the nervous and mental symptoms in soldiers. Strümpell, Nonne and others emphasized the important rôle played by psychogenic factors, while von Sarbó [48] warned that many so-called functional manifestations were due to organic "microcellular alterations."

The psychoanalysts stepped forth during the war and offered the result of more than a decade of intensive investigation to shed light on this controversial problem.[49] They insisted that too much emphasis was being placed on the physical and conscious mental factors. They demanded that more attention be paid to the possible rôle of affective and subconscious determinants, and they upbraided clinicians for neglecting and ignoring these and other less evident psychologic moments. Freud, Ferenczi and Abraham utilized this newer psychodynamic method in analyzing the behavior of injured soldiers. Stern, Mohr and Simmel used these principles in their therapeutic approach in such cases. These reactions are more than a "flight into illness" (Gaupp). The trauma situation endangers the integrity of the patient, is a blow to ego-security and results in regression to narcissism. There also arises an increased

45. Marinesco, G.: Lésions commotionelles expérimentales, Rev. neurol. **25**:329, 1918.

46. Mott, F. W.: War Neuroses and Shell Shock, London, H. Frowde, 1919.

47. Fuchs, A.: Zur Pathologie und Symptomatologie der Commotio Cerebri, Wien. med. Wchnschr. **77**:1229, 1927.

48. von Sarbó, A.: Granatenfernwirkungsfolgen und Kriegshysterie, Neurol. Centralbl. **36**:360, 1917.

49. Ferenczi, S.; Abraham, K.; Jones, E., et al.: War Neuroses and Psychoanalysis, London, International Psycho-Analytical Press, 1921.

ego-sensitiveness owing to damming up of libido, with resultant hypochondriasis. These investigators also emphasized that the difference in the response of persons to identical stresses lies not in constitutional peculiarities but in varying psychosexual experiences. Jones pointed out the importance of the conflict arising from the fact that unconscious impulses to cruelty are stimulated by war. Landauer [50] and Coriat,[51] among others, have applied these principles to the interpretation of the neuroses following trauma in civil life.

After the war there was a noteworthy reaction to the whole problem of the neuroses following trauma. A group of writers, mainly German, emphatically denied the existence of a traumatic neurosis. They firmly insisted that there is no such disease entity and strongly urged the acceptance of their theory that the psychologic reactions after an injury have no causal relation, strictly speaking, to the accident. Reichardt [52] was the outstanding defender of this new point of view. He was supported by investigators like Bonhoeffer,[53] Kleist [54] and Stier.[55] Such scientific agitation and polemic writing resulted in the refusal of the German courts, in 1928, to recognize these psychogenic reactions (Reichardt) following accidents as compensatable. Such extremism naturally called forth lively opposition. Frankel, Aschaffenberg,[56] Riese [57] and Honigmann [58] remonstrated and pointed out that whatever the exact pathogenesis the accident is a conditio sine qua non of the subsequent complaints.

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50. Landauer, Karl, in Piese, W.: Die Unfall-Neurose als Problem der Gegenwartsmedizin, Stuttgart, Hippokrates Verlag, 1929, p. 65.

51. Coriat, I. H.: Recent Conceptions of the So-Called Traumatic Neuroses, Boston M. & S. J. **195**:160 (July 22) 1926.

52. Reichardt, M.: Der heutige Stand der Beurteilung der sogenannten Unfallneurosen, Deutsche med. Wchnschr. **51**:213 (Feb. 10) 1928; Die psychogenen Reaktionen, einschliesslich der sogenannten Entschädigungsneurosen, Arch. f. Psychiat. **98**:1 (Nov. 22) 1932.

53. Bonhoeffer, K.: Beurteilung, Begutachtung und Rechtsprechung bei den sogenannten Unfallneurosen, Deutsche med. Wchnschr. **52**:179 (Jan. 29) 1926.

54. Kleist, K.: Zur Entschädigungsfrage bei den sogenannten Unfallneurosen, Klin. Wchnschr. **6**:1317 (July 9) 1927.

55. Stier, Ewald: Ueber die sogenannten Unfallneurosen, Leipzig, Georg Thieme, 1926.

56. Aschaffenberg: Zur Frage der psychogenen Reaktionen und der traumatischen Neurosen, Deutsche med. Wchnschr. **52**:1594 (Sept. 17) 1926.

57. Riese, W.: Traumatic Neurosis as a Problem of Contemporary Medicine, Stuttgart, Hippokrates-Verlag, 1929; reviewed, J. Nerv. & Ment. Dis. **75**:689, 1932.

58. Honigmann, George, in Riese, W.: Traumatic Neurosis as a Problem of Contemporary Medicine, Stuttgart, Hippokrates-Verlag, 1929; reviewed, J. Nerv. Ment. Dis. **76**:409 (Oct.) 1932.

HISTORY OF THE SEARCH FOR OBJECTIVITY

The recent tendency to deny compensation to persons considered to be neurotic forces to the foreground the problem of the differentiation of the neuroses following trauma from the results of organic injury to the intracranial contents. This now becomes an important and practical issue. The search for objective criteria proving injury to the nervous system is not new. As soon as the distinction between organic and functional disturbance became clear, in the last quarter of the nineteenth century, suggestions for practical ways of differentiation appeared in the literature.

Thomson and Oppenheim, in 1886, thought that anesthesia was a sign of organic disease. Oppenheim later accepted the teachings of the Parisian clinicians. Mankopf, in 1885, described acceleration of the pulse over tender points as a proof of organicity. Strauss,[59] in 1892, showed the limitations of this sign, while Rumpf accepted it. Rosenthal,[60] in 1897, could not confirm its value. Sachs,[61] in 1909, declared the sign useless. Guth [62] mentioned tachycardia and cardiovascular lability. Tilmann suggested cephalic hyperalgesia. Rumpf, in 1890,[63] added fibrillary twitchings and diminished response of muscles to galvanism. Other authors proposed hyperreflexia. Guth [62] and Sanger [64] mentioned changes in the visual fields as a valuable objective sign.

Not much progress could be made in this search for objective signs of intracranial injury as long as so much confusion reigned regarding the nature of the posttraumatic syndromes. So-called pathognomonic signs were constantly being described in nontraumatic conditions. The efforts of Friedmann, Neel, Tromner and others to separate out from this heterogeneous group the cases due to definite injury to neural tissues directed clinicians to attempt to find ways of demonstrating the existence of such damage to the intracranial contents. The accumulating information regarding what goes on after serious blows to the head, from the anatomic and physiologic standpoints, and the remarkable recent

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59. Strauss, A.: Ueber den Werth des Mannkopf'schen Symptomes bei Nervenleiden nach Trauma, Berl. klin. Wchnschr. **29**:1222 (Nov. 28) 1892.

60. Rosenthal, H.: Zur Charakteristik einiger "objectiven" Symptomen bei den sogenannten traumatishen Neurosen, Inaug. Dissert., Leipzig, 1897.

61. Sachs, Heinrich: Die Unfallneurose—ihre Entstehung, Beurteilung und Verhütung, Berlin, Preuss und Junger, 1909.

62. Guth, Georg: Ueber den diagnostischen Wert einzelner Symptome der traumatischen Neurose, Inaug. Dissert., Berlin, 1890.

63. Rumpf: Beiträge zur kritischen Symptomatologie der traumatischen Neurose, Deutsche med. Wchnschr. **16**:165 (Feb. 27) 1890.

64. Sanger: Diskussion über Nervenunfallerkrankungen, Neurol. Centralbl. **15**:509 (April 22) 1896.

additions to the armamentarium of the diagnostician now permit a fairly accurate determination of whether there are organic changes to account for the patient's complaints.

Before discussing this newer approach to the old problem, we wish to call attention to a number of relatively recent attempts to find evidence of organic injury in disturbances of vegetative innervation. These investigations arose from the recent widespread emphasis on the importance of vasomotor disturbances in cerebral concussion. The Muck [65] epinephrine test is the best known of these recent contributions. Its value is still debatable and will be determined by further confirmatory studies, such as that of Riecke.[66] The following signs have also been suggested: a change in the pulse rate when the patient is turned upside down (Knauer [67]); a difference in blood pressure between the two sides (Pal [68]); hyperemia of the tympanum (Muller [69]); capillaroscopic alterations (Goldbeck-Loewe [70]); changes in venous pressure (Villaret and Jonescu [71]). More clinical studies are needed to evaluate the significance of these signs.

THE COOPERATIVE INVESTIGATION

The application of the methods that we are about to describe permits more precisely than ever before the separation in a given patient of the organic and the psychogenic—the primary effects of the blow and the secondary psychic elaboration. We have gathered together from the work in the various departments the fruits of the special investigations and shall try to show how a more constant utilization of these valuable technics will help to bring some order into this rather chaotic and extremely vital subject. We contend that as long as these special studies are not made organic disease of the brain cannot be definitely excluded.

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65. Muck, O.: Ueber das Wesen und die klinische Bedeutung eines nasalen vasomotorischen Reflexphänomens, Ztschr. f. Hals- Nasen u. Ohrenh. **13**:311, 1925-1926; Die Begutachtung postkommotioneller Spätfolgen unter Berücksichtigung des Adrenalinsondenversuchs, Klin. Wchnschr. **10**:1713 (Sept. 12) 1931.

66. Riecke, H. G.: Ergebnisse mit dem Muckschen Adrenalin-Sonden-Versuch bei Schädel-Hirn-Unfallverletzten, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **30**:298 (Sept.) 1932.

67. Knauer, A.: Zur Erkennung und Begutachtung der Hirnerschütterung und ihrer Spätfolgen, Monatschr. f. Unfallh. **36**:337, 1929.

68. Pal, J.: Unnoted Symptom of Cerebral Trauma, Compt. rend. Cong. internat. de méd. trop. et d'hyg. **1-2**:624, 1928.

69. Muller, cited by Bremer et al.: J. de neurol. et de psychiat. **32**:466, 1932.

70. Goldbeck-Loewe: Ueber die Rolle der Kapillarmikroskopie bei der Beurteilung von angeblichen "traumatischen Neurosen," München. med. Wchnschr. **76**: 491, 1929.

71. Villaret, M., and Jonescu, D.: Les modifications de la pression veineuse au cours des hémiplégies organiques et des séquelles des traumatismes craniocérébraux, Presse méd. **34**:1265, 1926.

Neurologic Examination.—The importance of careful, exhaustive neurologic examination cannot be overemphasized. Since the war so much stress has been put on the psychologic and social factors in the nervous symptoms following accidents that the necessity for adequate neurologic examination has apparently been relegated to the background. Faulty diagnoses result, and there is repeated failure to discover the presence of focal involvement of the central nervous system. Focal lesions often are very small and give rise to clinical changes difficult to detect without careful investigation. Neustadt [72] recently summarized these difficulties by noting: the failure to detect the presence of organic disease as a result of inadequate neurologic examinations; the frequent lack of knowledge of neurologic semeiology even on the part of examiners who aspire to be thorough; the frequency of diagnostic prejudice after reading of the opinions of others, and the tendency to think along rigid, classificatory lines.

Goldstein [73] aptly showed that apparently normal and identical performances in a group of patients during a routine examination do not necessarily imply the operation of similar neuropsychologic processes. An aphasic person may conceal his defect by undue taciturnity, and an alexic patient, by associated writing movements not evident to the examiner (Goldstein and Gelb.[74]). Normal behavior and performance in clinical tests may be accompanied by a subjective feeling of difficulty and a consciousness of unusual strain. These reports should not be ignored. They are at times very significant (Piffl and Pötzl [75]).

Careful clinical studies, with attention to detail, will result in fewer cases with no evidence of focal involvement of the nervous system. Mann [76] recently enumerated certain neurologic findings which are not noted or the significance of which is often overlooked and which undoubtedly point to material damage to the brain. He mentioned difficulty in lateral conjugate gaze, a slight tendency to fall to one side, diminished corneal reflexes and slight disorders in coordinated movements. In 1915, Goldstein [73] urged that more attention be paid to the investigation of cerebellar signs and symptoms in patients with gunshot

72. Neustadt, R.: Zur Beurteilung psychischer Störungen bei Hirnverletzten, Nervenarzt **3**:141, 1930.

73. Goldstein, Kurt: Ueber den zerebellaren Symptomenkomplex in seiner Bedeutung für die Beurteilung von Schädelverletzten, München. med. Wchnschr. **62**:1439, 1915.

74. Goldstein, Kurt, and Gelb, A.: Psychologische Analysen hirnpathologischer Fälle auf Grund von Untersuchungen Hirnverletzter, Ztschr. f. d. ges. Neurol. u. Psychiat. **41**:1, 1918.

75. Piffl, O., and Pötzl, O.: Ein otogener parietaler Hirnabszess, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **112**:93, 1925.

76. Mann, L.: Ueber ein häufig zu beobachtendes Syndrom bei Commotio bzw. Contusio Cerebri, Deutsche med. Wchnsch. **57**:2172 (Dec. 25) 1931.

wounds of the head. He indicated that mild involvement of the cerebellar pathways is frequently overlooked and that the patients are considered neurotic. Reichman,[77] a year later, reported a number of such cases which were considered "traumatic neuroses." Residuals of aphasia may easily be missed, and unless the condition is thought of, mild forms of sensory agnosia may be overlooked. Schilder and Stengel[78] (one case of head injury) pointed out the importance of pain asymbolia, and Gerstmann,[79] Rad[80] and others have recently emphasized the clinical features of lesions in the parieto-occipital region of the brain. Changes in tonus limited to one limb, slight postural defects, defective associated movements, convergence reactions (Schilder[81]) and other less striking manifestations of disease of the basal ganglia are sometimes not noted during the usual neurologic examination, especially in compensation practice. These clinical signs cannot be ignored.

The evaluation of these clinical findings is extremely important. This is especially true of minimal changes, such as an isolated positive Rossolimo reflex, mild contraction of the visual fields or diminished vibratory sensation in the lower limbs. Kino[82] recently pointed out the importance of the depression of the tendon reflexes after head injuries. Katzenstein[83] discussed transitory changes in the shape of the pupils following head injury. The Philadelphia school of neurologists (Pearson[84]) is doing a valuable piece of work in determining the range of distribution of so-called positive neurologic signs in normal groups. Their future contributions may force us to modify some of our clinical interpretations. In one of our patients we noted a loss of associated movements in walking on one side. This fact was not even mentioned in the report by the other medical experts. When this omission was brought to the attention of these examiners they answered

77. Reichman: Die Bedeutung der funktionellen Kleinhirndiagnostik zur Beurteilung von Kopfschussverletzungen, Deutsche med. Wchnschr. **42**:9, 1916.

78. Schilder, Paul, and Stengel, Erwin: Das Krankheitsbild der Schmerzasymbolie, Ztschr. f. d. ges. Neurol. u. Psychiat. **129**:250, 1930.

79. Gerstmann, Joseph: Eine umschriebene Störung der Orientierung am eigenen Körper, Wien. klin. Wchnschr. **17**:1010, 1924.

80. Rad, C.: Kasuistische Beitrag zur Symptomatologie in der Uebergangsregion der Parietal und Occipitallappens, Ztschr. f. d. ges. Neurol. u. Psychiat. **131**:273, 1930.

81. Schilder, Paul: Clinical Note on the Convergence Reaction in Alcoholics, J. Nerv. & Ment. Dis. **71**:722, 1930.

82. Kino, F.: Reflexstörungen bei Erschütterungen des Zentralnervensystem, Deutsche Ztschr. f. Nervenh. **116**:49 (Dec.) 1930.

83. Katzenstein, Erich: Veränderungen an der Pupillenform bei Commotio et Contusio Cerebri, Schweiz. Arch. f. Neurol. u. Psychiat. **27**:286, 1931.

84. Pearson, G. H. J.: The Effect of Age on Vibratory Sensibility, Arch. Neurol. & Psychiat. **20**:482 (Sept.) 1928.

that the finding had no clinical significance. In a subsequent report this change was again deleted in spite of its accepted value in pointing to the existence of organic disease. Clinical observations should be recorded regardless of the physician's opinion of their significance at the time of examination. Vagueness of a clinical finding is no proof of its psychogenicity, nor is one's inability to explain or understand a sign or symptom cogent evidence that it is functional. Such experiences should spur one to further investigation.

The decision as to whether a particular syndrome is psychogenic or due to involvement of neural tissue is at times difficult. The careful studies of Kleist [85] and the observations of Claude [86] and others on the variants of psychomotor disorders in disease of the brain makes us less certain about calling psychogenic any motor disturbance which does not readily fit into a well recognized clinical pattern. The tendency of some examiners to ignore unfamiliar changes and unusual clinical findings by relegating them to the vague group of "functional" manifestations is pernicious and deserves mention in order to be condemned. Gumpertz [87] called attention to the occasional difficulty of differentiating between hysterical and true epileptic convulsions. We recently studied a patient whose condition on a previous admission to the hospital was considered psychogenic because of atypical seizures. He later returned with definite epileptic convulsions.

Case 1.—J. C., an Irish laborer, aged 39, on Aug. 2, 1929, was struck by a large piece of concrete which fell 21 feet (6.3 meters). He lost consciousness. He entered the Mount Sinai Hospital in the middle of October, 1931, complaining of attacks of dizziness and vomiting for fifteen months. Since the accident he had had peculiar attacks during which he suddenly experienced a weak feeling in the knees, a feeling of chilliness ran up his back and a gripping sensation occurred in the epigastrium. He then vomited, fell to the floor and lost consciousness. An attack lasted only a moment and was followed by a period of nervousness, during which he was incontinent.

On his first admission to the hospital one of these attacks was carefully observed and recorded as follows: "His body became rigid and his skin hyperemic, and his eyes began to bulge. He lay on his back for about thirty seconds in a position of opisthotonos. He uttered some guttural sounds and with a tremendous amount of energy whirled himself onto his abdomen. This movement was repeated several times. He was conscious and kept on saying 'My head hurts.'" There was no

85. Kleist, K.: Untersuchungen zur Kenntnis der psychomotorischen Bewegungsstörungen bei Geisteskranken, Lepizig, Dr. Werner Kleinkhardt, 1908.

86. Claude, H., and Baruk, H.: Les troubles psycho-moteurs d'origine cérébrale, hypertonie, mouvements automatiques et sommeil cataleptique, Presse méd. **39**:233 (Feb. 18) 1931.

87. Gumpertz, Karl: Ist die Reaktionstheorie der nach Unfällen auftretenden Nervenschädigungen aufrechtzugehalten? Deutsche Ztschr. f. Nervenh. **116**:176, 1930.

evidence of focal disease of the central nervous system. There was a bizarre sensory syndrome, and on the patient's discharge the opinion was that he was suffering from a traumatic neurosis.

On his second admission to the hospital in February, 1932, true convulsive attacks were noted, with clonic movements of the right upper and lower limbs. Encephalography showed the subcortical markings to be extremely prominent, especially in the parietal region. The ventricles were normal. The spinal fluid was under increased pressure.

This case illustrates the caution with which one must interpret the less typical motor, convulsive and dyskinetic syndromes following head injury. The presence of sensory changes functional in type should not lead one to assume that the whole reaction is psychogenic. The epidemic of encephalitis taught some memorable and valuable lessons. Since that time physicians have become less certain and dogmatic regarding the differentiation between the functional and the organic. The dystonic syndrome, for example, is frequently baffling to the clinician. Identically patterned movements are called now functional, now organic, depending on the history and on other details of the clinical setting. May [88] called posttraumatic compulsive tendencies psychogenic. Yet compulsive reactions occur in chronic encephalitis. Pick,[89] a number of years ago, noted them in cerebrovascular disease. That organic disease may lower thresholds and permit the emergence of abnormal behavior patterns or subconscious psychic constellations is accepted.

Psychologic and Psychiatric Survey.—The psychiatric examination is usually in the form of an interview and is limited to determination of the so-called mental status and to a study of the personality make-up of the patient. If there is no evident gain from illness a more careful study from the psychodynamic standpoint is sometimes attempted to assist in ascertaining the cause for the persistence of symptoms. There is often neither time nor equipment for undertaking such investigations. The neurosis following trauma may be a screen behind which the person hides, a flight from conscious and subconscious difficulties or a temporary solution to harrowing conflicts; or it may satisfy a pressing need for punishment. Indeed, the accident itself at times proves to be an attempt at suicide (Landauer).

We wish to draw attention especially to the need for an adequate survey of the conscious processes of those with head injuries. We refer to the careful study in a particular case of the nature of the intellectual processes, the quality of the sensory experiences and the status of the volitional tendencies. All these and other aspects of mental life can be

88. May, S.: Ueber psychogene Störungen bei Hirnbeschädingten, Ztschr. f. d. ges. Neurol. u. Psychiat. **75**:460, 1922.

89. Pick, A.: Die neurologische Forschungsrichtung in der Psychopathologie und andere Aufsätze, Abhandl. a. d. Neurol., 1921, pt. 13, p. 1.

studied accurately and even quantitatively. The precise description of these phenomena in posttraumatic states is not a mere academic pastime. The usual psychiatric examination is incomplete and will detect only the most evident defects.

The widespread neglect of the study of the conscious processes is, in part, due to the ready assumption that minimal psychic defects are functional. This attitude is based on the occasional similarity of these mental changes to those encountered in psychogenic disease. This resemblance becomes much less evident with more careful, critical clinical analysis.

There is no doubt in the mind of any one as to the organicity of the severe posttraumatic enfeeblement or constitution. Careful scrutiny of the mental changes in the less severe instances of head injury forces the conclusion that there is present in these cases an organic reaction syndrome. The ready fatigability, emotional lability, difficulty in thinking and mild symptoms of mental defects are similar to the change that one sometimes finds in the early stages of the organic psychoses, as in dementia paralytica, cerebral arteriosclerosis and even tumor of the brain (Hoch,[90] Bürger-Prinz,[91] Bunker,[92] Baruk[93]). No one questions the organicity of these subjective symptom complexes, even in the absence of focal phenomena. One must recognize that impairment of psychologic processes is just as important and significant of involvement of neural tissue as hemianopia is of disturbance of the optic pathways or centers.

A special technic is available for the investigation of conscious psychologic processes, which has been slowly developed since the days of Wundt. These methods have much to offer neuropsychiatrists if they will make use of them. Kraepelin early in his career realized the value of such methods and used them freely in his investigations.[94] Sommer[95] and Gregor[96] summarized the possibilities of the use of

90. Hoch, August: The Dementia of Cerebral Arteriosclerosis, Psychiat. Bull. New York State Hosp. **9**:306 (July) 1916.

91. Bürger-Prinz, Hans: Die beginnende Paralyse, in Foerster, O., and Willmanns, K.: Monographien aus dem Gesamtgebiete der Neurologie und Psychiatrie, no. 60, Berlin, Julius Springer, 1931.

92. Bunker, H. A.: The Very Early Symptoms of General Paresis, Am. J. M. Sc. **171**:386 (March) 1926.

93. Baruk, H.: Les troubles mentaux dans les tumeurs cérébrales, Thèse de Paris, no. 446, Paris, O. Doin, 1926.

94. Jelliffe, S. E.: Kraepelin, the Man and His Work, Arch. Neurol. & Psychat. **27**:761 (April) 1932.

95. Sommer, Robert: Lehrbuch der psychopathologischen Untersuchungsmethoden, Berlin, Urban & Schwarzenberg, 1899.

96. Gregor, Adalbert: Leitfaden der experimentellen Psychopathologie, Berlin, S. Karger, 1910.

experimental psychology in psychopathology. The application of these methods requires special training and, if possible, a laboratory. Goldstein [97] in his Institute of Brain Injuries in Frankfurt, Isserlin [98] in Münich, Poppelreuter [99] in Bonn and Fröschels [100] in Vienna have shown to what extent these methods can be applied to problems of clinical neurology. Wells [101] and Franz [102] have worked along these lines in clinical psychiatry. For the details of the methodology the publications of the investigators mentioned should be consulted. There are many sources of error in the gathering of the data and in the interpretation of the results. Poppelreuter [103] outlined these difficulties in great detail. These special studies at times yield information of great value. The more complete picture of the mental processes that results from this approach is of particular value because it suggests at times practical methods of rehabilitation for the injured (Fröschels, studies on aphasic patients).

The commonly used intelligence test, the Binet-Simon and the performance (Pintner) tests are of little value in many of these cases because of the absence of a previous record of performance. With the more widespread routine examination of students at all ages such controls may become available. Hardwick [104] recently reported the case of a boy who had previously been given such an examination. The comparison of his performance before the accident with that observed afterward yielded valuable information.

Isserlin studied disorders of memory in persons with head injuries, using the Ebbinghaus technic of learning and relearning nonsense syllables. Observations of the details of the learning process resulted at times in interesting and useful facts. He noted, for instance, certain

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97. Goldstein, K.: Die Behandlung, Fürsorge und Begutachtung der Hirnverletzten, Leipzig, F. C. W. Vogel, 1919.

98. Isserlin, M.: Ueber Störungen des Gedächtnisses bei Hirngeschädigten, Ztschr. f. d. ges. Neurol. u. Psychiat. **85**:84, 1923.

99. Poppelreuter, Walter: Ueber psychische Ausfallsercheinungen nach Hirnverletzungen, München. med. Wchnschr **62**:489 (April 6) 1915.

100. Fröschels, Emil: Die Kopfverletzungen im Kreige: Ihre psychologische Untersuchung, Behandlung und Fürsorge, Vienna, Moritz Perles, 1918.

101. Wells, F. L.: Mental Tests in Clinical Practice, New York, World Book Company, 1927; Psychometric Factors in Medical Problems, Am. J. Psychiat. **8**: 235 (Sept.) 1928.

102. Franz, S. I.: Handbook of Mental Examination Methods, New York, The Macmillan Company, 1919.

103. Poppelreuter, Walter: Psychologische Begutachtung der Erwerbsbeschränkten, in Abderhalden, E.: Handbuch der biologischen Arbeitsmethoden, Berlin, Urban & Schwarzenberg, 1928, sect. 6, pt. 6, no. 6. p. 369.

104. Hardwick, Rose S.: Intelligence Tests in a Case of Brain Injury, Psychol. Bull. **24**:185, 1927.

cases in which the patients could learn only with the help of visual images. He discovered in this way residuals of sensory aphasia. He analyzed the various types of difficulty in retaining and reproducing learned material and sought to determine from the particular reactions the cause for this defect in the learning process. It is important to know whether there is a real effacement of engrams or whether the poor performance is due to fatigue, vacillating attention or some other unfavorable condition. He claimed also to have isolated a functional, nonorganic pattern of response in the memory tests which differs from that found in organic cases. In this connection, the Wells memory test for psychotic patients should be mentioned.

The nature of the fatigability in posttraumatic cases has interested experimental psychologists intensely. They have used the Kraepelinian ergographic work curves, and have made dynamometric and reaction time experiments. The resulting curves have been analyzed and compared with those for normal controls. What is still available in the psyche—what can be salvaged—is estimated, and plans are made accordingly. A detailed summary of these contributions is not within the scope of this paper. The results of the work of these investigators as well as their methods of attack should be brought to the attention of clinical psychiatrists and neurologists.

It is informing to learn that these experimental methods borrowed from psychologic laboratories can occasionally be of surprising aid in the solution of clinical problems. We have already mentioned Isserlin's detection of residual aphasia with the Ebbinghaus technic. Goldstein and Gelb demonstrated the value of tachistoscopic studies in optic agnosia. They reported a remarkably detailed and accurate analysis of a case of alexia in a man with a head injury which was discovered during tachistoscopic experiments and was not evident in the ordinary reading tests. Busch [105] and Altenburger [106] reported the early detection by tachistoscopy of hemianopic defects in the fields in the absence of any defect for color or form. Busch detected a defect in spatial orientation in a man with an injury of the occipital lobe by his inability to perform during a maze test.

The day has passed when the neuropsychiatrist can ask the injured person a few questions and as a result of a brief interview form an opinion as to the intactness of all the psychologic processes, the patient's adaptive capacity and his personality make-up. A more careful analysis,

105. Busch, A.: Ueber die Ausfallserscheinungen nach Sehhirnverletzungen und einige Vorrichtungen zur Prüfung der optischen Orientierung und der Arbeitsanpassung, Ztschr. f. ang. Psychol. **19**:156, 1921.

106. Altenburger, H.: Die Aufdeckung hemianopischer Gesichtsfelddefekte durch die tachystoskopische Methode, Arch. f. Psychiat. **88**:477, 1929.

utilizing if possible these psychologic methods, is advised for doubtful cases. This, however, can never replace the personal impressions left by even a brief interview. Results of tests and exact quotients cannot take the place of the opinion of an experienced clinician.

Too much emphasis has been placed in the past on the necessity for the presence of intellectual defects in cases of organic disease of the brain. The experience with encephalitis has also been striking in this respect. The defect may be largely limited to the affective sphere. There may be no difficulty in solving simple arithmetical problems, but there may be impairment of judgment in the sense of inability to evaluate ethical and social problems, lack of earnest relation to reality, loss of interest and initiative and other defects in the volitional processes (Feuchtwanger [107]). Busch called attention to the defective adaptive capacity of those with head injuries—an inability to alter readily the tempo and rate of work. He also suggested practical ways of detecting such insufficiency. All these disorders of the mental life may be due to injury to neural pathways and centers, though the same defects are occasionally noted in psychogenic disease. Some of them are perhaps a result, as Friedmann has noted, of inability to mobilize psychic energy.

The Ophthalmologic Survey.—The extensive distribution of the visual pathways and the position of the visual centers in the occipital lobes expose them to involvement during head injury. McCullough [108] said that some degree of impairment of the visual fields is present in every case of head injury. Extensive defects in the fields may exist without any complaint. If careful studies are made in occipital and other injuries many interesting variations of field defects are found. This is illustrated by the rich and instructive literature dealing with experiences during the war (Inouye,[109] Beauvieux,[110] Morax, Moreau and Castelain [111] and others). Thorough investigations of the visual fields are not made often enough in the cases of diffuse blows to the head in civil life.

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107. Feuchtwanger, Erich: Die Funktionen des Stirnhirns—ihre Pathologie und Psychologie, in Foerster, O., and Willmanns, K.: Monographien aus dem Gesamtgebiete der Neurologie und Psychiatrie, no. 38, Berlin, Julius Springer, 1923.

108. McCullough, C. J.: Importance of Visual Field Studies After Head Injuries, Pennsylvania M. J. **33**:733 (Aug.) 1930.

109. Inouye, Tatsuji: Die Sehstörungen bei Schussverletzungen der kortikalen Sehsphäre. Nach Beobachtungen an Verwundeten der letzten japanisch Kriege, Leipzig, Wilhelm Engelmann, 1909.

110. Beauvieux: Les troubles visuels dans les blessures par coup de feu de la sphère visuelle corticale ou des radiations optiques, Arch. d'opht. **35**:472, 1917.

111. Morax, Moreau and Castelain: Les différents types d'altérations de la vision maculaire dans les lésions traumatiques occipitales, Ann. d'ocul. **156**:1 (Jan.) 1919.

De Schweinitz,[112] Mahillon [113] and others denied that field defects can be simulated even by trained observers. The color fields cannot be neglected. The defect may be limited to color perception. Since Posado-Armigo,[114] in 1884, described a case of color blindness following head injury, other similar, at times less complete, impairments of color perception have been reported (Worms [115]). Gelb,[116] in 1918, reported such a case, carefully analyzed from the psychologic point of view. Mitchell and de Schweinitz [117] have shown that constriction of the visual fields, even in functional cases, may be limited to color alone.

A correct technic in the study of the visual fields is important. Wolffberg [118] indicated the importance of proper, constant illumination. Nicoletti [119] spoke of negative results of perimetric studies with ordinary illumination and the detection of a ring scotoma with a reduced amount of light. The usual approximate tests made in office practice, especially by neurologists, are of little value for the detection of many of the field defects. Halbertsma [120] wrote of a patient who after a head injury showed merely bilateral concentric narrowing of the fields with the perimeter. After using the tangent screen, he found a relative central scotoma on one side and an absolute scotoma on the other side.

Any complaint of poor vision justifies a thorough investigation. Good visual acuity, negative results of field tests with the usual office procedures, normal fundi and the absence of refractive errors do not justify the assumption of the existence of psychogenic disease or simulation. To illustrate the importance of this point of view, the following case is reported.

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112. de Schweinitz, G. E.: The Relation of the Visual Fields to the Investigation of Certain Neuroses and Psychoses, Univ. Pennsylvania M. Bull. **22**:282, 1910.

113. Mahillon: Mise au point de la question de l'utilité du relevé des champs visuels colorés pour le diagnostique et le prognostic de certains troubles nerveux consécutifs aux traumatismes, Rec. d'opht. **26**:17, 1904.

114. Posado-Armigo, A.: Dyschromatopsie traumatique, Rec. d'opht. **6**:468, 1884.

115. Worms, G.: Les troubles visuels subjectifs chez les blessés craniocérébraux, Ann. d'ocul. **160**:456, 1923.

116. Gelb, Adhémar: Color Vision in Brain Injuries, Deutsche Ztschr. f. Nervenh. **59**:216, 1918.

117. Mitchell and de Schweinitz, cited by de Schweinitz.[111]

118. Wolffberg, L.: The Important Clinical Points in Perimetry with Special Reference to Traumatic Neurosis, Arch. Ophth. **33**:597, 1904.

119. Nicoletti, G.: Sul comportamento e significato clinico della scotoma anulare nelle affezioni delle membrane interni dell'occhio e del nervo ottico, Ann. di ottal. e clin. ocul. **54**:874, 1926.

120. Halbertsma, K. T. A.: Ueber Gesichtsfeldstörungen nach Schädeltrauma, Klin. Monatsbl. f. Augenh. **78**:779, 1927.

CASE 2.—P. L., a woman, aged 19, while going to work on March 8, 1932, was struck by a truck; she was hurled into the air and fell to the pavement, striking her head. She was unconscious for a few minutes and then remained dazed for about five or six minutes. When she was seen by one of us on the next day she complained of severe headache, especially on movement of the head, and of generalized aches and pains. There was no external evidence of injury to the head. There was a left hemihypesthesia for all forms of sensation, of organic pattern. A Babinski sign was elicited on the left and there was right lower facial weakness. The patient complained of diminished vision in the left eye. In the hospital on the next day no sign of focal disease was found except vague sensory impairment in the left lower limb. The ankle jerk was somewhat more active on the left side. We were informed by the physicians at the hospital in which she was examined that the visual difficulty was probably functional, as all the investigations by ophthalmologists gave negative results.

Two weeks after her discharge from the hospital she still complained of diminished vision in the left eye. She saw objects as "through a cloud." Visual acuity as tested with the Snellen chart was normal and showed no change from the condition shown by a test in July, 1931. The fundi were normal; ocular tension was normal on palpation; there were no ocular palsies, and the pupils reacted well to light and in accommodation. The visual fields on the Peters perimeter were normal for form and somewhat restricted for color, though still within normal limits. Study of the fields with a Duane screen showed a well defined ring scotoma on the left. The scotoma could not be demonstrated with the ordinary perimeter, even with the smallest test object. Examination of the fields two weeks after the discovery of this defect showed that the annular scotoma was beginning to break up into islets of absence of vision. This field defect was for both color and form.

The scotoma in case 2 explained the complaints of "foggy" vision in the left eye. Subsequent cases of head injury were studied with the tangent screen, and we soon encountered another case of annular scotoma. This was rather surprising in view of the alleged rarity of this phenomenon, especially after head injury.

CASE 3.—G. L., a colored chauffeur, married, aged 29, complained of headache for fifteen months. He gave a history of gonorrhea, contracted three weeks before admission. Fifteen months before admission his taxicab collided with a large truck. He was taken to Bellevue Hospital while unconscious. There was bleeding from the nose at the time, and extensive ecchymosis was observed about the left eye. Soon after the accident he began to complain of occipital headaches. He remained in bed thirty-seven days. The headaches continued until his admission to the Mount Sinai Hospital in April, 1932. The pain was worse on mental or physical effort. He also continued to have recurring dizzy spells, "spots before the eyes" and occasional diplopia, and when he looked at lights they "would have the appearance of stars." He became somewhat more irritable and noted that he became frightened very readily.

Physical examination revealed acute gonorrhea, and a urethral smear was positive for gonococci. The left lobe of the prostate was enlarged. Neurologic examination gave negative results. The tendon reflexes were all hypo-active. The spinal fluid pressure was 80 mm. of water, and the protein content of the fluid was 30 mg. per hundred cubic centimeters. The intra-ocular tension was 16 mm.

in both eyes. The Bailliart ophthalmometric test showed a diastolic pressure of 53 mm. in the right eye and 54 mm. in the left. The general blood pressure was 105 systolic and 65 diastolic. Bilateral ring scotomas were found with the tangent screen (fig. 1).

The clinical studies could not be completed because of the acute gonorrhea. A reexamination about one month after the field studies described showed the fields to be greatly contracted. The narrowing of the fields was so marked that it overlapped the site of the annular scotomas.

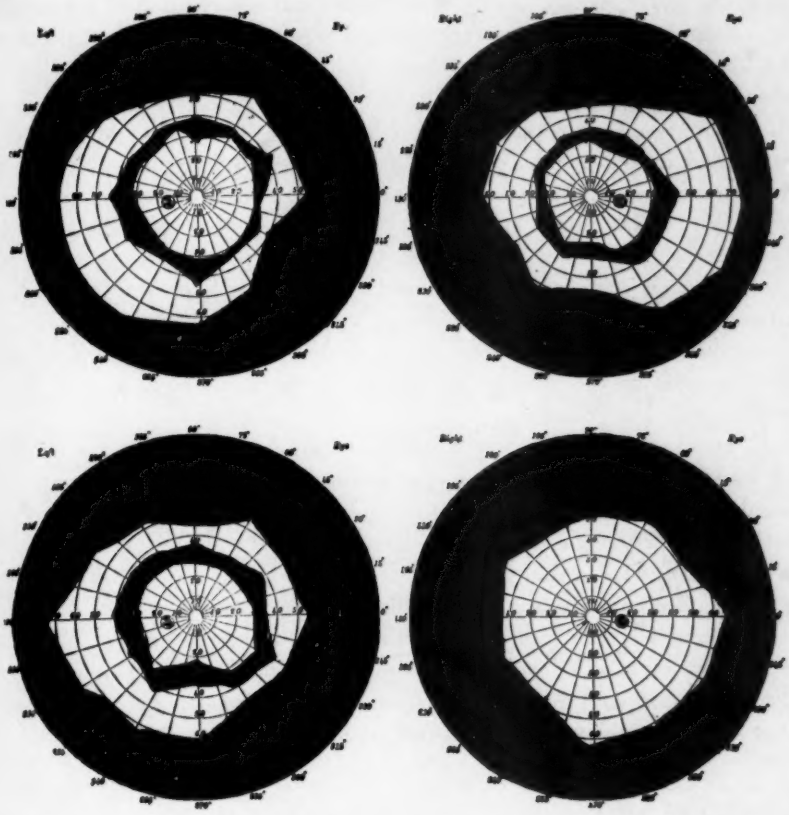

Fig. 1.—Visual fields of the patient in case 2 (below), taken on March 18, 1932, showing unilateral ring scotoma, and of the patient in case 3 (above), taken on April 15, 1932, showing bilateral ring scotoma. In both cases the tests were made in daylight and the patient showed good cooperation.

Cases 2 and 3 illustrate strikingly the principle that cases of head injury merit more thorough clinical investigation and emphasize the importance of unearthing the existent defects. The interpretation of the changes is another question. There is much to suggest the existence of a functional factor in case 3.

Although de Schweinitz, Wölfflin [121] and others have noted ring scotomas in cases of hysteria, Nicoletti,[119] reviewing thirty-three cases of annular scotoma seen in nine years among ten thousand cases of diseases of the eye, did not refer to hysteria but mentioned as etiologic factors retinitis pigmentosa, syphilitic chorioretinitis, myopia, injury to the eye, glaucoma and optic neuritis. Langdon,[122] in a recent review, did not mention hysteria, but added that ring scotomas have been reported in cases of solar retinitis, migraine, nasal sinus disease and commotio retinae and that they have developed as a result of looking into a furnace. Loddons,[123] Leblond,[124] Beauvieux [110] and others have reported this phenomenon in cases of head injury. Inouye [109] recorded it in a case of severe gunshot wound of the head. He somewhat dogmatically attributed it to hysteria, despite the fact that his patient had evidence of extensive focal disease of the brain. Gelb and Goldstein [125] reported six such cases after head injury, one of which was hemianopic and crescentic. They stated that the ring phenomenon is not hysterical but a manifestation of ocular fatigue. Pascheff [126] described ring scotomas in five cases of gunshot wounds of the occipital region and assumed them to be sequelae of cerebral injuries. The details of the pathogenesis of this interesting change in the fields have not yet been worked out.

The suggestion that ring scotomas may be due to fatigue is worthy of closer scrutiny, as fatigue and ready exhaustion run through the whole clinical picture of the postconcussion states. The various so-called fatigue curves reported in ophthalmologic literature—Foerster's shift type, Wilbrandt's exhaustion curve and the exhaustion spiral of von Reuss—need further study, and the conditions under which they are to be obtained need rigid standardization. Such information, obtained with perimetry, is of value, as a prominent mode of expression of a diseased brain may be ready fatigue. It is not reasonable to maintain that the only mechanism of this fatigue is ideogenous.

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121. Wölfflin, E.: Ueber ein seltenes Gesichtsfeldsymptom bei Hysterie, Arch. f. Augenh. **65**:309, 1910.

122. Langdon, H. N.: Ring Scotoma, in Contributions to Ophthalmic Science Dedicated to Dr. Edward Jackson, Menasha, Wis., George Banta Publishing Company, 1926, p. 274.

123. Loddons, G.: Sur un cas de scotome annulaire par la quinine, Ann. d'ocul. **166**:733, 1929.

124. Leblond, Etienne: Un cas de scotome annulaire bilatéral d'origine traumatique, Ann. d'ocul. **161**:740, 1924.

125. Gelb, A., and Goldstein, K.: Psychologische Analysen hirnpathologischer Fälle auf Grund von Untersuchungen Hirnverletzter: VII. Ueber Gesichtsfeldbefunde bei abnormer "Ermüdbarkeit" des Auges, Arch. f. Ophth. **109**:387, 1922.

126. Pascheff, C.: Die zerebralen Ringskotome und seltenere nervöse Augenstörungen nach Kriegsverletzungen, Arch. f. Augenh. **91**:233, 1922.

Much importance is sometimes attached to the constriction of the visual fields as an expression of functional disease. The persistent tubular contractions are usually hysterical. The lesser degrees of narrowing of the fields may be due to a great many causes. Contraction of the visual fields and color reversal have been observed in nitrobenzene poisoning, in chronic alcoholism and in poisoning with tobacco and with lead. Cushing and Bordley noted such constriction in cases of increased intracranial pressure. Goldstein [127] stated that constricted fields may be due to organic disease of the brain.

Bailliart's [128] recent description of ophthalmometry, a study of the diastolic pressure within the retinal arteries, suggests an interesting and perhaps valuable aid in the study of the state of the intracerebral circulation. The increased retinal diastolic pressure is alleged to appear before other signs of increased intracranial pressure. It is also said that these changes in retinal arterial pressure are an expression of the instability of the cerebral vasomotor system, a not uncommon sequel of head trauma. Lewy [129] has applied ophthalmometry to the study of chronic cerebral adhesive cystic arachnoiditis and has incorporated the findings in his delineation of the syndrome. This procedure has hardly been applied in cases of head injury and merits further study. Worms,[115] Claude and his co-workers [130] and Bremer and his associates [131] have published observations along this line. We have begun to use this method and have found an increased diastolic retinal pressure in almost all the cases with definite evidence of intracranial damage.

Bizarre visual experiences are not to be considered lightly, and no conclusions regarding their significance should be recorded without careful clinical studies (optic alloesthesia, Herrmann and Potzl;[132] dysmorphopsia, Gelb and Goldstein;[133] defective spatial orientation, Busch,[105] Feuchtwanger [107] and others). Seeing is not merely the pro-

127. Goldstein, K.: Visual Fields, Deutsche Ztschr. f. Nervenh. **59**:199, 1918.

128. Bailliart, P.: La circulation rétinienne dans les états d'hypertension intracranienne, Ann. d'ocul. **159**:785, 1922.

129. Lewy, F. H.: Der Adhäsionskopfschmerz als Folge der Meningitis serosa adhaesiva circumscripta, Ztschr. f. klin. Med. **116**:36, 1931.

130. Claude, H.; Lamache, A., and Dubar, J.: Le liquide céphalorachidien dans les séquelles de traumatismes craniens san fracture, Paris méd. **1**:271 (March 24) 1928.

131. Bremer, F.; Coppez, H.; Hicquet, G., and Martin, P.: Le syndrome commotionel tardif dans les traumatismes fermés du crâne, J. de neurol. et de psychiat. **32**:466 (July) 1932.

132. Herrmann, and Potzl, O.: Die optische Allaesthesie. Studien zur Psychopathologie der Raumbildung, Abhandl. a. d. Neurol., 1928, pt. 47.

133. Gelb, A., and Goldstein, K.: Psychologische Analysen hirnpathologischer Fälle auf Grund von Untersuchungen Hirnverletzter: IX. Ueber eine eigenartige Sehstörung ("Dysmorphopsie") infolge von Gesichtsfeldeinigung, Psychol. Forsch. **4**:38, 1923.

jection of retinal images on the cortex. These sensory experiences are elaborated by the visual cortex into perceptions. Disorder of this mechanism results in various forms of psychic blindness, or optic agnosia. The defects may be slight and may escape notice without close observation. The patients are sometimes considered peculiar and even psychotic because of their strange behavior, clumsiness, lack of insight and absence of awareness of their own defects. Poppelreuter [99] recorded such a case, in which the condition was treated as a psychosis. This absence of the consciousness of defect is noteworthy, and its significance was emphasized some time ago by Anton.[134] The clinical changes that we are interested in at this moment are often minimal and not striking. Head blows frequently cause fine, small lesions, with consequent less evident clinical alterations. Poppelreuter, for instance, noted one case in which an optic agnostic defect became evident only after the patient looked at moving pictures. This same soldier was able to read and describe fairly complex pictures. Occasionally there may be difficulty in manipulating objects, though ability to recognize them is retained—optic apraxia. Poppelreuter described a case of literal alexia—preservation of ability to read printed matter with inability to recognize the individual letters. We recently observed a case of alexia following a head injury.

Case 4.—T. P., a German fireman, aged 45, was struck by an automobile on March 9, 1931, and was unconscious for two days. Following the accident he complained of headaches and dizziness, which were relieved a number of times by lumbar tap. He also discovered that he could not read or recognize letters. He was able to read before the accident. There was also a history of convulsive seizures and other less clearly defined attacks of loss of consciousness. He was observed at the Mount Sinai Hospital during April and May, 1932.

Physical examination showed scars on the face, ears and upper limbs, sequelae of a burn sustained in the explosion of an oil tank in 1905. The blood pressure was 150 systolic and 106 diastolic; the heart was enlarged on the left. Neurologic examination revealed pure alexia, with no other focal signs. There was no hemianopia at a number of examinations. There was no agraphia. Slight dysgraphia was present but could not be evaluated because of the absence of knowledge of the patient's previous chirography. He copied well from dictation but could not read his own handwriting. Body orientation was intact, and there was no finger agnosia. He was able to read numbers. There was a marked reading defect for words and for letters. No other type of optic agnosia was present. There was no evidence of any residual of sensory or motor aphasia. Mimicry was good. No dyspractic phenomena were present. An examination a few months later showed, in addition to the findings mentioned, a well marked hemisensory syndrome for all modalities of sensation of a functional pattern, including involvement of all the special senses on the same side.

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134. Anton, G.: Ueber die Selbstwahrnehmung der Herderkrankungen der Gehirns durch den Kranken bei Rindentaubheit und Rindenblindheit, Arch. f. Psychiat. **32**:86, 1899.

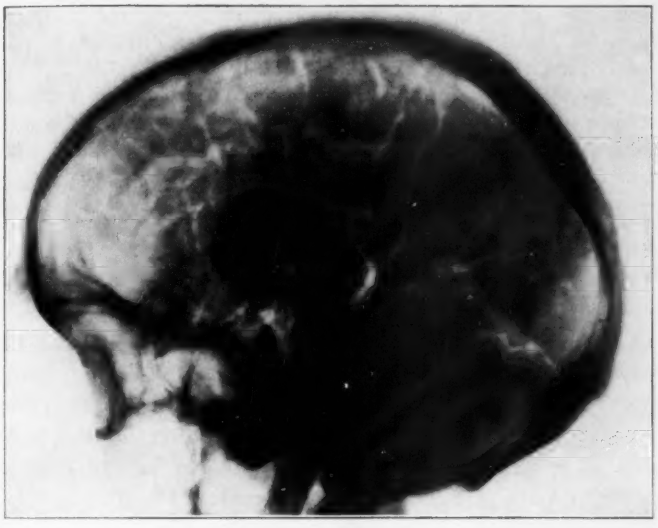

Fig. 2 (case 4).—Encephalogram showing an increased amount of air over the frontal and parietal lobes and also over the occipital region.

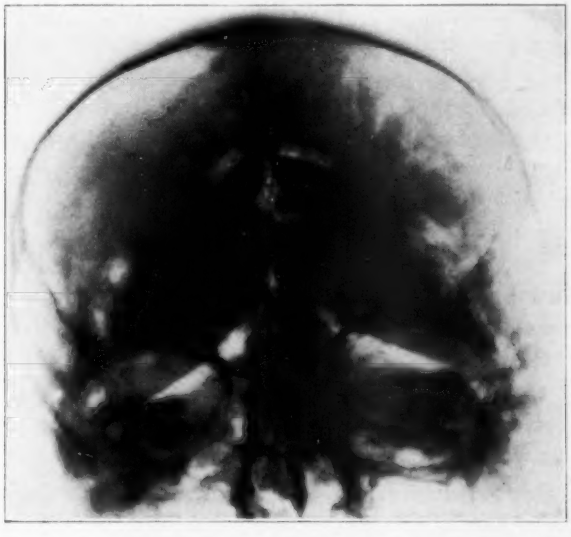

Fig. 3 (case 4).—Encephalogram showing failure of the ventricles to fill, dilatation of the subarachnoid spaces of both hemispheres, especially the left, and an increased amount of air subtentorially.

Examination of the spinal fluid, including determination of the pressure, gave results within normal limits. The Wassermann reactions of the blood and of the spinal fluid were negative. An encephalogram showed a well defined collection of air over the cortex. The ventricles did not fill.

Early in October, 1932, a favorable settlement in regard to compensation was made. Examination on Dec. 4, 1932, showed the persistence of the alexia in a pure form as already described. The ability to recognize letters was a trifle better. The patient had been making efforts to regain his power to read, but he could not recognize words. The sensory changes on the right side of the body were less marked, and the functional deafness in the right ear was gone.

Alexia following head injury is not common. Massary [135] recently gave an excellent comprehensive review of the subject. Alexia of organic origin as the sole focal sign is indeed unusual. Right hemianopia or its residuals are practically the rule, though Lannois and Tournier,[136] Fröschels [137] and Schuster [138] reported cases without any changes in the fields. Even these authors reported hemiparesis and aphasia in their cases. A small lesion can conceivably involve the pathways between the angular gyrus and the left occipital cortex (lingual and fusiform lobes) and spare the adjacent optic radiations. A lesion of the angular gyrus itself results in more than pure alexia (agraphia).

The associated sensory changes suggest the possibility that in case 4 the alexia was hysterical. The absence of neighborhood phenomena and of other focal changes supports this hypothesis. The clinical picture in this case resembles, with remarkable accuracy, the so-called pure alexia of Dejerine—ability to write but not to read letters and words and conservation of the ability to read numbers. We think that such a dissociation favors the existence of organic disease to account for the alexia. If the condition were simulated or functional we should expect a more global defect. The occurrence of epileptiform seizures favors the existence of an organic disease of the brain. The encephalographic picture, showing a localized collection of air over the cortex, strengthens the theory of an organic pathogenesis of the alexia. If the entire syndrome is psychogenic it is most unusual and unique in our experience. When the patient was last examined the alexia had persisted practically unchanged for about twenty-two months in spite of the fact that the undoubted psychogenic manifestations were improving rapidly. The epileptiform seizures continued in spite of the satisfactory money settlement.

Otoneurologic Investigation.—A thorough otoneurologic study is important. The watch test and simple clinical tests for labyrinthine func-

135. de Massary, J.: L'alexie, Encéphale **37**:53, 1932.

136. Lannois and Tournier: Cécité verbale sans cécité littérale et sans hémianopsie, J. de méd. de Paris **8**:117, 1896.

137. Fröschels,[99] p. 69.

138. Schuster: Ein neuer Fall von Alexie, Neurol. Centralbl. **25**:628, 1906.

tion are inadequate. Vestibular tests should be made, with accurate determinations of auditory acuity. It is important, in this type of investigation especially, to rule out if possible involvement of the ear and labyrinth before the accident.

Brunner,[139] as a result of his clinical observations and experimental work, noted how frequently the cochlear mechanism may be spared in the presence of demonstrable involvement of the vestibular apparatus after injuries to the head. He found involvement of the eighth nerve in 43 per cent of sixty cases of head injury. The frequency of degenerative lesions in the vestibular and cochlear nuclei in his experimental animals is noteworthy.

All persons with a history of head injury, no matter how atypical the description of their experiences (dizziness, etc.), deserve an objective search for derangement of the equilibratory apparatus. Mygind[140] correctly pointed out that a pathognomonic posttraumatic vertiginous syndrome does not exist. Lithicum and Rand[141] found abnormal responses to vestibular tests in all of the thirty-six patients who complained of posttraumatic dizziness. Five of these patients were neurologically normal. Their observations led them to conclude that such manifestations are probably due to a combination of central and organic injury. Though the subjective symptoms disappear after a while because of compensation, the abnormal reactions to the vestibular tests persist. Barré and Greinier[142] described twenty cases in which there were no complaints and no clinical evidence of a disorder of the vestibular mechanism and yet definitely abnormal responses to vestibular tests were obtained. Groves[143] and others noted that the severity of the initial injury has little to do with the gravity of the subsequent otologic findings.

By using the audiometer, Groves found some degree of deafness in thirty-one of forty-two patients with head injuries. Twelve of the patients might easily have passed for persons with normal hearing. These patients showed so-called patchy tone gaps. With more normal controls for the distribution of islets of deafness this type of investigation of the cochlear mechanism may give valuable information.

139. Brunner, Hans: Pathologie und Klinik der Erkrankungen des Innenohres nach stumpfen Schädeltraumen, Monatschr. f. Ohrenh. **59**:696, 1925.

140. Mygind, S. H.: Traumatic Vestibular Diseases, Acta oto-laryng. **1**:515, 1918.

141. Lithicum, F. H., and Rand, C. W.: Neuro-Otological Observations in Concussion of the Brain, Arch. Otolaryng. **13**:785, 1931.

142. Barré, J. A., and Greinier, G.: Troubles vestibulaires chez les traumatismes craniens, Rev. d'oto-neuro-opht. **10**:633 (Nov.) 1932.

143. Groves, W. E.: Otologic Observations in Head Trauma, Arch. Otolaryng. **8**:249 (Sept.) 1928.

Brunner [139] and Bleyl [144] noted apparent hearing defects owing to the fact that the patient was examined when fatigued.

Study of the Spinal Fluid (Including Encephalography).—1. Spinal Fluid Pressure: Head injuries often give rise to disturbances of the cerebrospinal fluid mechanism. Investigation of the spinal fluid and of the cerebrospinal spaces has become an indispensable aid in the approach to the problem of the sequelae of head injury. Defective absorption, and sometimes overproduction, of cerebrospinal fluid (Gielen [145]) and frequent interference with the free flow of the fluid result in alterations in intracranial and spinal fluid pressure. There is much cogent evidence pointing to the probability that the subjective syndrome following head injury may be caused, at least in part, by these changes.

The precise mechanism of these disorders is not clear. In addition to the disorganization of the resorptive surface by meningeal adhesions and thickening, circulatory and vasomotor defects may play a significant rôle. Why does one not see such late and persistent sequelae more regularly after spontaneous subarachnoid bleeding? Schlecht [146] expressed the belief that the increased production of spinal fluid is due to a vasomotor disorder within the choroid plexus. Such a mechanism is likely in our case 17, in which the spinal pressure was 360 mm. of water and bilateral papilledema appeared very soon after the head trauma, although the spinal fluid was clear and normal.

Cannon,[147] in 1901, demonstrated experimentally an increase in intracranial pressure immediately following trauma to the head. Quincke,[148] in 1910, told of eight cases in which the only objective evidence of organic injury in posttraumatic states was increased spinal pressure.[148a] Seventeen years before he had noted that chronic serous meningitis may be the cause of persistent headaches and dizziness and in this way give rise to a neurosis-like syndrome. In 1921, Kron [149]

144. Bleyl, R.: Die Beeinflussung der Hörprüfung durch die Ermüdung des Hörnerven, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:62, 1932.

145. Gielen: Meningitis serosa cystica nach Kopftrauma, Nervenarzt **1**:487, 1928.

146. Schlecht, H.: Zur Frage der Meningitis serosa traumatica, Deutsche Ztschr. f. Nervenh. **47-48**:697, 1913.

147. Cannon, W. B.: Cerebral Pressure Following Trauma, Am. J. Physiol. **6**:91, 1901.

148. Quincke, H.: Kopftrauma und Spinaldruck, Monatschr. f. Unfallh. **17**:388, 1910.

148a. We recently observed in the Morrisania Hospital a case in which severe headaches (paroxysmal) and dizziness of two months' duration after head injury were completely relieved by lumbar puncture. The spinal pressure was over 300 mm. of water.

149. Kron, J.: Meningitis serosa traumatica (circumscripta et diffusa), Ztschr. f. d. ges. Neurol. u. Psychiat. **69**:34, 1921.

commented on the neurasthenia-like clinical pictures in circumscribed serous meningitis after head trauma and noted as the only objective finding hypertension of the cerebrospinal fluid. Gerhartz [150] more recently recorded increased spinal fluid pressure in six patients as the only evidence of intracranial damage. The spinal fluids were otherwise normal. He emphasized that these patients were unjustly considered simulators and advised that one make manometric studies before expressing such an opinion.

Because of extensive meningeal adhesions, the manometric reading of the spinal fluid pressure may be normal in spite of the actual presence of increased pressure (Payr [151]). Not enough emphasis has been placed in the past on the significance of very low manometric readings. We have recently observed two cases in which patients with varied complaints following head injury showed improvement after encephalography in which the spinal fluid pressure reading rose after the insufflation. The experiences in the following case were striking and instructive. The initial pressure reading was 60 mm.; following the injection of 35 cc. of air and marked improvement subjectively the reading ten days later was 140 mm. Case 8 showed a similar low manometric reading (60 mm.).

Case 5.—N. W., a watchmaker, aged 48, was admitted to the Mount Sinai Hospital on Feb. 26, 1932. He complained of nervousness and stated that for the past three years he had had almost constant headache, with attacks of vertigo and periods of unconsciousness at night. He had been nervous and fearful all his life. He gave a history of an injury to the head twelve years before, which produced unconsciousness for about ten minutes. He complained of occasional headache and dizziness before the onset of the present symptoms three years before, and also of a tremor of the fingers which prevented him from working.

On examination there were found: a perforated nasal septum; ankylosis of the left elbow secondary to an old injury, and left lower facial weakness. The left knee and ankle jerks were more active than those on the right, and the left abdominal reflexes were less active. The patient feared that he was "going insane." His condition was considered as anxiety hysteria by one observer and as psychopathic personality by another. To most of those who studied the patient in the wards, the whole reaction seemed psychogenic, except for the reflex changes noted.

The spinal fluid pressure was 60 mm. on March 1, 1932. The fluid was otherwise normal. Thirty-five cubic centimeters of air was injected into the subarachnoid space; no more air could be injected, for no more fluid could be obtained. No encephalograms were taken. On March 11, another spinal tap and manometric study were done. The pressure on that day was 140 mm.

Immediately following the injection of air the headaches and dizziness disappeared; the anxiety also disappeared. The patient stated that he had not felt so

150. Gerhartz, H.: Lumbaldrucksteigerung als Spätfolge von Schädelverletzten, Med. Klin. **25**:1194 (Aug. 9) 1929.

151. Payr, E.: Meningitis serosa bei und nach Schädelverletzungen (traumatica), Med. Klin. **12**:841 (Aug. 6) 1916.

well for a long time. He regained facility in the use of his fingers. However, he insisted on leaving the hospital, and further studies could not be made. He was still free from headache and dizziness six months after the injection of air.

The injection of air in this case unquestionably broke up meningeal adhesions somewhere in the cerebrospinal spaces, releasing something which interfered with the circulation of the cerebrospinal fluid.

2. Chemistry of the Spinal Fluid: An increased protein content in the spinal fluid is indicative of involvement of the meninges and, in certain instances, of the neural parenchyma. Demme,[152] using Kaffka and Samson's technic, recorded seven cases in which an increase in spinal fluid protein was the only evidence of organic intracranial involvement following head injury. These abnormal values may sink to normal. The change in the protein content may be the only alteration in the spinal fluid.

Henschen [153] suggested the value of chemical studies of the fluid after trauma. He contended that continued breaking down of brain lipoids and proteins results in an accumulation of organic acids in the spinal fluid and even in the urine. Studies of the state of the hemato-encephalic barrier of meningeal permeability may prove of considerable interest (Hoff).

3. Encephalography: Since the work of Foerster and his associates,[154] in 1926, encephalography has become an invaluable adjuvant in the investigation of cases of head injury. We think that the method has already proved its worth. The few feeble attempts to minimize its value and significance are not convincing. The criticisms of Reichardt and his school are not valid. These investigators have not offered any encephalographic data of their own. They have emphasized the psychogenic reactions in posttraumatic states, something which we find almost all the patients show if they are followed long enough and continue to be socio-economically maladjusted.

No experienced clinician relies solely on encephalographic findings. One must evaluate the pictures in the setting of the whole clinical complex, including the history. A history of encephalitis, convulsions or retarded development before the accident should make one extremely cautious in the interpretation of the encephalograms. The similarity of the encephalographic records in traumatic cases is striking and

152. Demme, Hans: Eiweissbefunde im Liquor von Schädeltraumatikern. Ein Beitrag zur Frage der Begutachtung von Kopfverletzten, Med. Klin. **30**:590 (April 17) 1930.

153. Henschen, C.: Ueber die Ursachen des postkommotionellen und postkontusionellen Hirndruckes, insbesondere über Hirnödem, Hirnschwellung und Hirnverkleinerung nach Schädelverletzungen, Zentralbl. f. Chir. **54**:3169, 1927.

154. Foerster, O.: Commotionsneurose, Beitr. z. klin. Chir. **137**:647, 1926. Foerster, O., and Penfield, W.: The Structural Basis of Traumatic Epilepsy and the Results of Radical Operation, Brain **53**:99, 1930.

cannot be considered accidental. Schwab,[155] Hauptmann,[156] Friedman,[157] Penfield [158] and many others have already admitted this method as a necessary addition to diagnostic technic. Reports are now rapidly coming in from all over the world proving the organicity in many of the cases of the mental changes following head injury, or at least the existence of an organic nucleus around which a particular reaction has developed. Despite the absence of normal controls, the impression of most workers, including ourselves, is that physiologic variants of the contour of the ventricles and the subarachnoid spaces are rare and that, given a good technic, certain well known changes on the x-ray plates indubitably point to the existence of some degree of previous and present intracranial damage.

The encephalographic findings often cannot account for all the complaints. The pictures give limited but valuable information. They permit one sometimes to gage the degree of cerebral damage or at least to convince oneself that organic changes took place. Lack of filling and unequal filling of the ventricles, increased surface markings, extensive localized collections of air over the cortex and pulling of the ventricles to the side of the injury are all important and significant of intracranial damage. These distortions are secondary to the original injury. The presence of such changes seems to be closely correlated with the existence of the subjective posttraumatic syndrome. That marked clinical symptoms may be present without encephalographic changes and that profound alterations revealed by such studies may exist without any complaint is not surprising. Apfelburg,[159] Feuchtwanger,[107] Poppelreuter [103] and others have reported extensive injuries of the frontal lobe without any clinical manifestations. There are undoubtedly other important factors, circulatory (vasomotor) and perhaps chemical, which cannot be visualized in the roentgenogram and which are significant in the understanding of real causes for the subjective symptom complex.

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155. Schwab, O.: Encephalographie, Liquorpassage und Liquorresorptions—Prüfungen im Dienste der Beurteilung von sogenannten Commotionsneurosen, Ztschr. f. d. ges. Neurol. u. Psychiat. **102**:294, 1926.

156. Hauptmann, A.: Die Objektivierung postkommotioneller Beschwerden durch das Encephalogramm, Zentralbl. f. d. ges. Neurol. u. Psychiat. **48**:846, 1927-1928.

157. Friedman, E. D.: Effects and Appraisal of Head Injuries: III. Encephalographic Observations, Arch. Neurol. & Psychiat. **27**:791 (April) 1932.

158. Penfield, W.: Chronic Meningeal (Post-Traumatic) Headache and Its Specific Treatment by Lumbar Insufflation, Surg., Gynec. & Obst. **45**:747, 1927.

159. Apfelburg, B.: Gunshot Wound of Frontal Lobes Without Symptoms, U. S. Vet. Bur. M. Bull. **6**:863, 1930.

The unique value of this procedure lies in the relative ease with which intracranial alterations can be demonstrated in the absence of any of the usual signs of focal damage to the brain. Cases previously considered entirely functional are now being reopened. We have recently encountered a rather large number of such cases. At times we were astounded by the extent of the encephalographic changes. The cases of patients previously in the hospital and regarded as suffering only from psychogenic reactions are being reinvestigated whenever possible. Case 1 is such a case.

The patient who is neurologically entirely normal, whose roentgenogram shows no fracture of the skull and who presents only a series of subjective complaints is of particular interest. To complicate the problem, there is often present a sensory syndrome which is definitely psychogenic. In addition there may be a history of convulsive states. Matters are somewhat simplified if a seizure is observed. If no convulsion is seen during the period of observation, the encephalogram may be of considerable value.

Case 6.—M. M., an Italian laborer, aged 48, was admitted to the Mount Sinai Hospital on April 7, 1933, with the history that on Nov. 30, 1931, he fell 13 feet (4 meters), striking his head on a concrete floor. He lost consciousness for eleven days. On awakening he complained of pain in the left side of the head and neck, which was made worse by sneezing, coughing, straining and change of position. He remained in the hospital one month. Since discharge, his complaints had been headache, dizziness, tearing of the eyes, twitching of the left side of the face and of the left hand, and attacks of loss of consciousness with foaming at the mouth but no incontinence.

Neurologic examination showed: bilateral exophthalmos; poor mimetic facial innervation on the left side; diminished pain, touch and temperature sensation involving the left side of the scalp, face, neck and chest down to the nipple line and also involving the entire left upper extremity; impairment of vibration sense in the left upper extremity, and diminished hearing on the left side. All these sensory changes were considered to be psychogenic. The patient was cooperative and well oriented, showed no memory or other intellectual defects and had good insight into his condition.

Encephalography was done, and 230 cc. of clear, normal fluid, not under increased pressure, was removed; 170 cc. of air was introduced in the usual way. Roentgen examination of the skull after the injection of air showed a symmetrical dilatation of the entire ventricular system. There was no displacement of the ventricles, and their outlines were normal. There was moderate dilatation of the basilar cisterns. The cortical markings were exaggerated in the right frontoparietal region, and there was noted in this region a small collection of air over the cortex, suggesting underlying atrophy.

To complete the clinical studies, the following tests were made:

1. The Muck epinephrine test. Both turbinates were painted with a 1:1,000 solution of epinephrine. The left turbinate remained blanched for about thirty-five minutes, and the right for fifteen minutes. The reaction was somewhat more diffi-

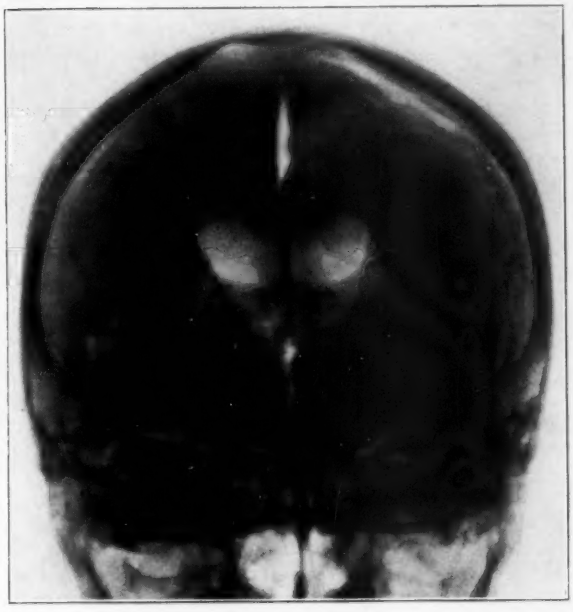

Fig. 4 (case 6).—Encephalogram showing internal hydrocephalus and an increased amount of air over the left cerebral cortex.

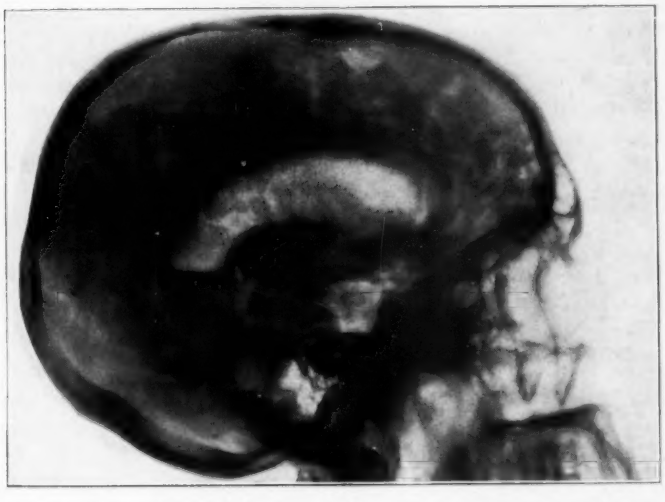

Fig. 5 (case 6).—Encephalogram showing internal hydrocephalus and an increase in the amount of air over the cerebral cortex.

cult to evaluate on the right side because the turbinate was much less vascular before the epinephrine was applied. The reaction was positive.

2. The Bailliart ophthalmometric test. The average tension of the retinal arteries in three readings was: diastolic, 65 mm., and systolic, 115 mm. The intraocular tension was: right, 16 mm., and left, 17 mm. The blood pressure in the right arm was 130 systolic and 80 diastolic, and in the left arm, 150 systolic and 90 diastolic. These readings are rather high.

3. A test for resorption of spinal fluid. Two cubic centimeters of 10 per cent sodium iodide was injected intrathecally. Specimens of urine were collected for forty-eight hours. No iodine was recovered in any of the specimens. This is suggestive of defective resorption of cerebrospinal fluid.

The additional tests were merely confirmatory. The various results of the special tests confirm the presence of definite organic intracerebral damage. We recently studied another similar case in which there were no focal signs on clinical examination, but a convulsive state and striking encephalographic changes were present.

CASE 7.—M. K., a shipping clerk, fell down two flights of stairs on the morning of June 14, 1930. He recalled nothing until he found himself in the Beekman Street Hospital that afternoon. He was delirious for eleven days. Roentgen examination of the skull showed no evidence of fracture. He was told that he had numerous convulsive attacks in the hospital. He remained in the hospital for three weeks. The first convulsive attack after discharge was on a day toward the end of October, 1930; a second one occurred on Jan. 1, 1931, and a third, toward the end of March, 1931. He was seen for the first time on May 6, 1931. He did not complain of headache, dizziness or any of the other usual posttraumatic symptoms.

Examination on May 6 revealed no evidence of any organic disease of the central or the peripheral nervous system or of internal organs. Examination at the Mount Sinai Hospital in January, 1933, showed: the right pupil was larger than the left, and neither reacted fully to light stimulation; the right palpebral fissure was wider than the left.

Encephalography showed considerable dilatation of the lateral and third ventricles and a collection of air in the left parietal region, which extended from the frontal lobe back almost to the occipital region.

The absence of any subjective symptoms in this case in spite of the extensive encephalographic alterations is interesting and emphasizes the fact there are factors other than those made visible by this valuable procedure that are responsible for the subjective syndrome. There is no harm or danger in the procedure if the proper technic is used. It is interesting to note that reports are accumulating as to the therapeutic value of encephalography. Penfield [158] advised it for the treatment of posttraumatic headache. Lewy, Friedemann,[160] von Sarbó [161] and others

160. Friedemann, A.: Unerwartete Heilwirkung nach Hirnluftfüllung, Ztschr. f. d. ges. Neurol. u. Psychiat. **138**:440, 1932.

161. von Sarbó: Die mikrostruckturellen traumatischen Veränderungen des Nervensystems im Lichte der Kriegserfahrungen, Schweiz. Arch. f. Neurol. u. Psychiat. **29**:127, 1932.

Fig. 6 (case 7).—Encephalogram showing internal hydrocephalus.

Fig. 7 (case 7).—Encephalogram showing dilatation of the lateral ventricles.

reported unexpected recoveries following insufflation of air. We recently encountered a similar case. The man actually returned and requested that the encephalography be repeated. We have already referred to the complete and persistent relief of symptoms following encephalography in case 5.

Case 8.—L. D., a painter, aged 38, was admitted to the Mount Sinai Hospital on Oct. 14, 1932, with a history of headaches for eight years. In 1924 he fell from a fourth story window and was unconscious for about three days. Since then he had suffered from headaches, especially in the left parietal region. He was not able to sleep at times because of the headache.

Fig. 8 (case 8).—Encephalogram showing slight displacement of the ventricular system to the left. The left ventricle is larger than the right, and there is dilatation of the whole ventricular system.

Physical examination showed a defect of the skull in the left frontoparietal region, vitiligo, a healed fracture of the left clavicle and a perforated septum (history of a nasal operation). On this admission no definite signs of focal involvement of the nervous system were found. Mentally the patient was irritable and seclusive, at times mildly depressed and at other times unusually happy. There was no evidence of impairment of the intellectual processes.

The spinal fluid pressure was 60 mm.; the total protein was 35 mg. per hundred cubic centimeters; Wassermann tests of the blood and spinal fluid were negative. Encephalography was attempted, but was unsuccessful, as the spinal fluid ceased to flow after 30 cc. of air was injected. The headaches were much better following this procedure. The Muck epinephrine test was positive on the left side.

The patient was readmitted on Dec. 27, 1932, from the outpatient department. He was referred back for repetition of encephalography because of the distinctly favorable effect of this procedure on the subjective symptoms. The patient requested the insufflation.

On the second admission two defects were noted on the left side of the skull, one of them probably postoperative. He continued to complain of headache and was still seclusive and emotionally unstable. In addition, a slight difference in the size of the pupils was found, and there was some limitation of extreme lateral gaze to the right. The right knee and ankle jerks were hyperactive.

Encephalography showed an inequality in the ventricular system, which was dilated; the left lateral ventricle was larger than the right. There was a slight displacement of the ventricular system to the left.

Analysis of this case shows the following evidence that definite intracranial change was the basis of the complaints: low spinal pressure, with failure to obtain more than 30 cc. of fluid; subsequent normal pressure; relief of symptoms following encephalography; a distinctly positive Muck test on the side of injury; right-sided hyperreflexia, and encephalographic changes.

4. Resorption of Cerebrospinal Fluid: Increased spinal pressure with widening of the subarachnoid spaces is considered as due in part to defective absorption of cerebrospinal fluid. In Foerster's clinic in Breslau, this phenomenon has been studied by injecting 2 cc. of 10 per cent sodium iodide intrathecally and noting the time of its recovery from the urine. A marked delay in the appearance of iodine in the urine or failure to recover any trace of it after a given time is said to be indicative of defective resorption of cerebrospinal fluid. Phenolsulphonphthalein has also been used. Bielschowsky [162] and others have confirmed the value of this procedure. It deserves further trial and study. We have not been able to confirm the value of the sodium iodide test.

Comment.—Before the complaints of a patient with a history of a blow to the head can be dismissed as insignificant or psychogenic, he should be subjected to a cooperative investigation such as we have described. Absence of focal signs is no justification for dispensing with this procedure. Stern,[163] among others, recently urged such an approach to the problem of evaluation of the nature of head injuries.

We shall now refer to another case investigated in this manner. The patient was injured, and his claims for compensation were rejected because no significant sequelae were conceded to be present. The case was reopened after the completion of our investigation. He was granted

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162. Bielschowsky, P.: Störungen des Liquorsystems bei Schädeltraumen, Ztschr. f. d. ges. Neurol. u. Psychiat. **117**:55, 1928.

163. Stern, F.: Ueber die Spätfolgen von Gehirnerschütterungen und Kontusionen, Chirurg **4**:1, 1932.

some money on the basis of the existence of partial disability after the results of our clinical investigations were presented.

Case 9.—M. A., an Italian laborer, aged 42, whose past history was unimportant except for gonorrhea six years before admission, was admitted to the Mount Sinai Hospital on Feb. 15, 1932, with a history of dizziness since an injury to the head on June 25, 1931. His head was struck as he passed under a low window. He fell to the floor and was unconscious for about ten minutes. Soon after the accident he began to complain of dizziness and bilateral tinnitus. There was a scalp wound on the right side after the accident, and there was bleeding from both ears. He remained in bed for three months, complaining of intermittent pounding headache, vertigo, tinnitus and diminished hearing. He complained of frequent flashes of light before his eyes. He was up and about for four months before admission but found it impossible to resume his work because of marked dizziness on any change in the position of his body. He also complained of some weakness in the left lower extremity.

Physical examination showed: blood pressure, 110 systolic and 80 diastolic; a scar in the right parietal region; mild right lower facial weakness; a sluggish pupillary reaction to light, especially on the left; hyperactive tendon reflexes but no pathologic reflexes, and a definite impairment of the associated movement of the left upper extremity in walking. Careful psychologic examination was not possible because of the patient's illiteracy and because of poor rapport arising from linguistic difficulty. He had to be examined through an interpreter. There seemed to be no gross mental changes.

Caloric tests showed a delayed reaction on both sides, especially to stimulation of the horizontal canals. There was some nerve deafness on both sides, especially on the right. The otologist concluded that the right vestibular nerve was less responsive than the left. The intra-ocular tension was 18 mm. on both sides. A Bailliart ophthalmometric test showed a diastolic retinal arterial pressure of 60 mm. on the right and 62 mm. on the left. This was not a normal reading, especially when its relation to the systemic blood pressure is considered. Sodium iodide, 10 per cent, was injected intrathecally. No iodine was recovered in the urine after twelve hours. The Muck epinephrine test was positive on the right side. The Wassermann tests of the blood and spinal fluid were negative. The spinal fluid, including the pressure, was normal. An encephalogram showed very slight dilatation of the ventricular system, with no displacement; the third and fourth ventricles were not visualized. The subtentorial air was slightly exaggerated. The subcortical markings were not unusually prominent.

We did not consider the encephalographic changes pathologic. The few findings on the plates were within the normal range. Careful neurologic examination showed significant organic signs. The Muck test was positive on the injured side. The patient in case 8 also showed this reaction clearly on the side of the injury. The diastolic retinal pressure readings were high. This is the type of case in which complete examination as outlined must be made before a final decision is recorded.

Such a routine attack is time-consuming and expensive. It is not suggested that all cases of head injury should be studied in this manner. It is not necessary in the presence of evident gross focal signs and

symptoms (aphasia, hemiparesis, hemianopia). All cases in which the patient is apparently normal neurologically and in which the clinical picture is dominated by the usual subjective complaints of the posttraumatic state should be studied with these newer methods before a final opinion is given. The presence of psychogenic features in a given case is no reason for not resorting to this more accurate analysis. A wider use of this plan will do much to bring order into this important and hitherto rather confusing subject. Something will have to be done along these lines in the near future to minimize the number of unjust rewards and unfair rejections of claims for compensation. There is an urgent need for some plan to weed out the increasing number of fraudulent claims and to do away with the harsh injustice frequently meted out to the injured.

THE POSTCONCUSSION SYNDROME: THE QUESTION OF ITS ORGANICITY

The implicit confidence and the rigid finality of some observers who assert that most of the persistent complaints of the postconcussion state are psychogenic and result from the subtle suggestions of environment and from unconscious wishes for security are at times surprising. On the other hand, there is much to support the contention that the subjective complaints in many cases are due to organic changes within the skull. The same complaints (headaches, dizziness, fatigability, etc.) are encountered in those unconcerned with the alluring fruits of a favorable decision by a group of experts. It is informing, for example, to read the description by physicians of their own experiences following head injuries. Such relatively well trained observers, though before their injuries strong believers in the psychogenic nature of the multiple complaints of patients with head injuries, readily become converted to the theory that these clinical changes are often due to organic disease of the brain.

Durand-Weaver,[164] after a head injury, wrote that she doubted the importance of the desire for compensation as a cause of the postconcussion phenomena. Mayer[165] suffered severe headaches and dizziness after almost every movement for half a year following a severe blow. He urged his medical readers to believe their patients with head injuries when they complain. His symptoms persisted after the insurance and compensation aspects of his case were satisfactorily settled. He denied that the symptoms had a psychogenic basis. One of us was consulted by a physician following a head injury. The report of his case follows:

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164. Durand-Weaver, M.: Die Commotio Cerebri und ihre Bewertung, München. med. Wchnschr. **76**:1879, 1929.

165. Mayer: Commotio Cerebri und ihre Bewertung, München. med. Wchnschr. **76**:2135, 1929.

Case 10.—On Dec. 12, 1930, the patient gave the history that an automobile in which he was a passenger turned over on June 20, 1930. The window was open, and he was sitting beside the chauffeur. The right side of his head bumped against the curb. He was not unconscious at the time. He sustained an abrasion over the right ear. He continued with his medical duties and paid no further attention to the accident. Three days later he felt pain on the top of the head and some dizziness. The headache and dizziness then came on paroxysmally.

A few days after the onset of these symptoms he did not feel well, and remained miserable for a few days. He had to take ¼ grain (0.016 Gm.) of morphine to get rest. He was not able to leave his bed for two days. There was no nausea or vomiting. The headache and dizziness became constant. He did not fall at any time, but he had the sensation of falling, frequently having to hold on to something in order to steady himself. He returned to work after a few days of rest at home but found it impossible to continue. He left for a vacation in the Adirondacks, where he remained for four weeks. During the entire vacation period, though out of bed, he felt miserable and "groggy" and suffered from headaches. He returned home and to work at the end of July. He found, however, that he was unable to concentrate. He made an effort to take care of patients, but any exertion in that direction or any attention to the clerical details of his work would bring on headache. He was examined by an ophthalmologist in August, 1930, who found a smaller disk in the right eye than in the left and better vision in this eye. He received no other medical attention. The results of our physical and neurologic examinations were negative.

On Jan. 12, 1932, he wrote to one of us: "It is indeed a pleasure to inform you that I have fully recovered from the effects of injuries sustained in the auto accident of June, 1930. All my symptoms disappeared in about five months."

The suffering of these physicians probably arose as a result of actual changes within the cranial cavity. We have seen a few patients recently who presented such a subjective syndrome following trauma without any desire for compensation or a pending suit for damages. The resemblance of the complaints of these patients of various ages, of different personality organization and with dissimilar psychosexual backgrounds is worthy of note. The only common element in the cases is the history of an injury to the head. The assumption of the existence of unconscious determinants for the symptoms in the absence of evident motivation is not justifiable. The rarity of an identical syndrome in cases of psychoneurosis points to the existence of another factor, undoubtedly related to the head trauma, which is responsible for the particular symptom complex. Even the determination of an adequate cause for flight into illness does not nullify the causal rôle of the injury to the head. We believe that the pattern of the whole subjective syndrome bespeaks its organicity. We shall show later that psychogenic factors can and often do complicate the clinical picture.

Case 11.—A boy, aged 7 years, fell off the back of a truck and was unconscious for a short time. Immediately following the injury he began to complain of headaches and dizziness, especially on change of position. These complaints were continuous for about six months. He was unable to go to school. Any attempt to

focus his attention or to read resulted in severe headache. The symptoms recurred paroxysmally for about four years. There was never any suit. The accident was a result of the boy's own negligence. His adjustment in school was good to the date of the accident.

CASE 12.—A schoolgirl, aged 14, was seen on Nov. 22, 1932, complaining of dizzy spells and headaches of two weeks' duration. The complaints set in after she bumped her head against a wall as she tried to find her way through the room in the dark. There was no loss of consciousness. There were no changes in behavior immediately after the injury. She continued to play on the street. Two days later she began to complain of attacks of dizziness. Between the attacks she told of vague pains in the head, not localized in one area. There was marked tenderness of the head, especially on "the top" during the attacks of vertigo. On a few occasions the attacks lasted a long time—on one occasion a whole day. She also reported occasional headache and exacerbation of the dizziness on bending.

Neurologic and physical examinations gave entirely negative results. Examination of the spinal fluid, including the manometric reading, gave negative results. The Wassermann test of the blood was negative. The past history threw no additional light on the cause for the headaches and dizziness.

Tromner,[41] among others, has reported such complaints following trauma in children. The clinical pictures hardly differ from those in injured working men. There is no concept of possibility of gain from the illness. We recently examined a young woman struck by thugs who presented a similar problem.

CASE 13.—R. C., a single woman, aged 24, was struck on the right side of the head by thugs, presumably with a black-jack. She was unconscious for about ten minutes. A roentgenogram of the skull showed a fissured fracture in the right temporoparietal region. She was seen by one of us about five hours after the assault.

Examination showed mild confusion, left lower facial weakness and a right hemisensory syndrome, functional in pattern. There was ecchymosis over the right supra-orbital region. She was first seen on Jan. 10, 1932.

She was examined again on July 2, 1932. She had remained in bed for three weeks and did not go to work till one week after she left the bed. Since she was hurt she had had headaches and dizziness. She would become very dizzy on attempting to swim. Looking at moving pictures would cause an intense headache. There was frequent nausea but no vomiting. For one month following the injury she was unable to read because of the dizziness and headaches resulting from such activity. The headaches were more intense during rainy weather. She continued to work as a stenographer in spite of her discomfort.

There was a history of peculiar seizures antedating the injury. She would faint, and occasionally there was incontinence of urine at the time. There was no mention of convulsive movements. One such seizure was observed by a physician, who informed us that in his opinion the "fainting spell" was a "neurosis." The details of the seizures also suggested hysteria.

We were therefore dealing with a hysterical person who received a blow on the head and sustained a concussion of the brain. The hysterical sensory disorder was evident immediately after the injury. The subjective complaints had persisted, though somewhat less intensely, for a year.

This young woman was not insured and could not claim compensation for the injury. There was no question of any type of litigation. She had a good position. In this case one must separate the functional from the organic. There is evidence of probable hysteria antedating the injury. There is the possibility that if prospects of compensation had been present psychologic factors would have entered to interfere with her continued adjustment. There would have been no incentive to work in spite of discomfort. The subjective syndrome was also present in this instance without any obvious prospect of gain through illness.

Organic changes are not necessarily irreversible reactions; von Sarbó [161] recently pointed out the fallacy of assuming psychogenicity because of recovery. The intracranial alterations may be functional in the physiologic sense. A great deal has been written and much thought has been spent on the mechanism of concussion. The upheaval following a severe injury to the head has been conceived of in various ways. The truth probably lies in the cooperation of a concert of factors.

It will be well to review briefly the various opinions as to what goes on within the skull during and after a significant head injury and to indicate what forces are operative in bringing about the intracranial changes following commotio cerebri. The physical factors mentioned are as follows: compression of the cerebral cortex (von Bergmann [166]); pressure on the cortex, forcing out the blood —cerebral anemia (Kocher [167]); compression of the medulla oblongata (Breslauer [168]); compression of the whole brain stem (Schück,[169] Wilson and Winkelman [170]); shaking up of the cerebral centers plus anemia (Polis [171]); mass movements of the brain (Gussenbauer [172]); mass

166. von Bergmann, E.: Die Lehre von dem Kopfverletzungen, in Billroth and Luecke: Deutsche Chirurgie, Stuttgart, Ferdinand Enke, 1880, pt. 30.

167. Kocher, T.: Theorie des Hirndruckes nach Experimenten unserer Mitarbeiter und eigenen Untersuchungen, Hirnerschütterung, Hirndruck und chirurgische Eingriffe bei Hirnkrankheiten, in Nothnagel, H.: Specielle Pathologie und Therapie, Vienna, A. Hölder, 1901, vol. 1, pt. 3, p. 259.

168. Breslauer, F.: Zur Frage des Hirndrucks, Arch. f. klin. Chir. **103**:478, 1914.

169. Schück, F.: Hirnverletzung und Bewusstseinsfrage, Arch. f. klin. Chir. **167**:322, 1931.

170. Wilson, George, and Winkelman, N. W.: Gross Pontile Bleeding in Traumatic and Nontraumatic Cerebral Lesions, Arch. Neurol. & Psychiat. **15**:455 (April) 1926.

171. Polis, A.: Recherches expérimentales sur la commotion cérébrale, Rev. de chir., Paris **14**:644, 1894.

172. Gussenbauer: Die traumatischen Verletzungen, Stuttgart, Ferdinand Enke, 1880, p. 25.

movements of the spinal fluid with tearing of tissue (Duret,[173] Cassasa,[174] Berner [175]); disruption of intracranial elements owing to the varying elasticity of the constituents of the brain (Marinesco [45]); disturbance of the intracellular equilibrium as a result of differences in the specific gravity of cellular constituents (Rahm,[176] Ingvar [177]); tissue changes resulting from a difference in specific gravity between the gray and the white matter (Tilman [178]); mechanical agitation of the intracranial contents, resulting in molecular disorganization of the myelin sheaths (Russel [179]); a similar process resulting in molecular disorganization of the ganglion cells (Erichson,[15] Obersteiner [20]).

The physiologic factors that have been suggested are: vasospasms progressing to the point of softening of tissues (Neuberger [180]); reflex vasomotor paralysis (Hoff [181]); reflex spasm of the vessels of the choroid plexus, with diminished production of cerebrospinal fluid (Leriche [182]); vasomotor disorders in the choroid plexus, with increased production of spinal fluid (Schlecht [146]); vasomotor changes (stasis, prestasis), with hemorrhage (Ricker [183]); changes in the hematoencephalic barrier or meningeal permeability (Hauptmann [156]); defective resorption of cerebrospinal fluid (Fay [184]); breakdown of the

173. Duret, H.: Etudes expérimentales sur les traumatismes cérébraux, Thèse de Paris, 1878, no. 63.

174. Cassasa, C. S. B.: Multiple Traumatic Cerebral Hemorrhages, Proc. New York Path. Soc. **24**:101, 1924.

175. Berner, O.: Ueber kleine aber tödlich verlaufende traumatische Gehirnblutungen, die sogenannten "Dureschen Läsionen." Eine rechtsmedizinische Studie, Virchows Arch. f. path. Anat. **277**:386, 1930.

176. Rahm: Physikalische Beobachtungen zur Lehre von der Commotio Cerebri. Die Mechanik der Gehirnerschütterung, Beitr. z. klin. Chir. **119**:318, 1920.

177. Ingvar, Sven: Centrifugation of the Nervous System: An Investigation of Cellular Changes in Commotion, Arch. Neurol. & Psychiat. **10**:267 (Aug.) 1923.

178. Tilman: Die Theorie der Gehirn und Rückenmarkserschütterung, Arch. f. klin. Chir. **59**:236, 1899.

179. Russel, W. R.: Cerebral Involvement in Head Injury: A Study Based on the Examination of 200 Cases, Brain **55**:549, 1932.

180. Neuberger, Karl: Ueber zentrale traumatische Hirnerweichung und verwandte Prozesse, Deutsche Ztschr. f. d. ges. gerichtl. Med. **14**:383 (Feb. 15) 1930.

181. Hoff, H.: Experimentelle Studien zur Frage des postkommotionellen Hirnödems, Ztschr. f. d. ges. Neurol. u. Psychiat. **129**:583, 1930.

182. Leriche, René: De l'hypotension du liquide céphalo-rachidien dans les traumatismes du crâne, Presse méd. **39**:945 (June 27) 1931.

183. Ricker, G.: Die Entstehung der pathologisch-anatomischen Befunde nach Hirnerschütterung in Abhängigkeit vom Gefässnervensystem des Hirnes, Virchows Arch. f. path. Anat. **226**:180, 1919.

184. Fay, Temple: Generalized Pressure Atrophy of the Brain Secondary to Traumatic and Pathologic Involvement of the Pacchionian Bodies, J. A. M. A. **94**:245 (Jan. 25) 1930.

ependymal barrier and penetration of cerebrospinal fluid into the cerebral tissues (Hoff [181]); unresolved contusion (Trotter [185]); late cerebral edema (Constantini [186]); perversion of the activity of intracellular proteolytic ferments (Marinesco [45]); alteration in the irritability of the ganglion cells (von Sarbó [161]); continued microglial activity (Stevenson [187]); liberation of histolytic ferments as a result of the breakdown of brain tissue (Staemmler [188]); deprivation of oxygen resulting from vascular lesions, causing the tissues to take up water and swell (Cannon [147]); the effect of cytotoxic substances resulting from the breakdown of injured brain tissue (Joannovics [189]); changes in the acidity of the cortical cells (Knauer and Enderlen [190]); binding of water in cells—*Hirnschwellung* (Reichardt [191]); organic acids in the intercellular tissue of the brain due to breakdown of brain tissue causing edema (Henschen [153]); disorders of the vegetative nerve supply to the head (Muck [65]); changes in the activity of reticular tissue about blood vessels (Rossi [192]).

In addition to causing direct injury to brain structures, the intracranial disturbances mentioned result in softenings, hemorrhages, formation of scar tissue, atrophy, dilatation of the ventricles, an excessive amount of fluid over the brain, meningeal adhesions and other anatomic changes found in the brains of injured persons. Such tissue alterations may take place long after the original trauma. They do not always end in serious and irreparable defects. Many of these perversions of activity are reversible processes. Vasospasms may relax; the vasomotor irritability may disappear, and the excessive production of cerebrospinal fluid may cease. A number of the changes are, however, not imme-

185. Trotter, Wilfred: On Certain Minor Injuries of the Brain, Lancet **1**:935, 1924.

186. Constantini, H.: Traumatisme cranien. Commotion hypertrophique de Duret. Au 10e jour large trépanation décompressive. Guérison, Bull. et mém. Soc. de chirurgiens de Paris **47**:938, 1921.

187. Stevenson, L. D.: Head Injuries: Effects and Their Appraisal: II. The Rôle of the Microglia, Arch. Neurol. & Psychiat. **27**:784 (April) 1932.

188. Staemmler, M.: Ueber Veränderungen der kleinen Hirngefässe in apoplektischen und traumatischen Erweichungsherden und ihre Beziehungen zur traumatischen Spätapoplexie, Beitr. z. path. Anat. u. z. allg. Path. **77**:409, 1927.

189. Joannovics, G.: Zur Wirkung fermentativ gewonnenen Spaltungsprodukte aus Geweben und Bakterien, Wien. klin. Wchnschr. **33**:648 (July 22) 1920.

190. Knauer, A., and Enderlen, E.: Die pathologische Physiologie der Hirnerschütterung nebst Bemerkungen über verwandte Zustände, J. f. Psychol. u. Neurol. **29**:1 (Nov.) 1922.

191. Reichardt, M.: Hirnschwellung, Allg. Ztschr. f. Psychiat. **75**:34, 1919.

192. Rossi, O.: Linee fondamentali di traumatologia del sistema nervoso centrale, Riv. di pat. nerv. **38**:797, 1931.

diately reversible. They may persist, as has been shown by Ricker for vasomotor instability. The restoration of equilibrium takes time—from a few months to a number of years.

Since the first attempt to study the problem of concussion experimentally by Gama,[193] in 1835, there have been many attempts to attack the subject in this way. They all tend to justify the operation of most of the factors mentioned. Jakob's [194] monograph summarized the literature up to 1913. He observed degeneration of tracts, involvement of the ganglion cells, foci of softening and small hemorrhages. The changes were most marked in the cervical region of the cord and in the bulb. Earlier investigators, as well as Gama, did not use animals. They experimented with fluids of varying consistencies simulating the contents of the skull (Alquié, Fischer and Ferrari [195]). Felizet [195] used dead skulls filled with paraffin. Pirogoff,[196] in 1864, first used experimental animals. Various lesions were described, and ingenious theories were spun from the experimental data as to how the physical blow to the skull affected the intracranial contents. Jakob's [194] and Schmaus' [197] work merely confirmed the well known fact that ganglion cells may suffer directly from the blow. Recently, Hashiguchi [198] noted dilatation of the ventricles after hammering the heads of young dogs. Schonbauer,[199] on the other hand, utilizing the encephalographic technic in animals, noted swelling of the brain and diminution of the size of the ventricles. Such studies add little to the data gathered in the ordinary clinicopathologic correlation. Little additional light was thrown on the problems of pathogenesis. However, the careful work of Knauer and Enderlen [190] deserves praise in that it proves rather cogently the important rôle that disturbance of the cerebral vasomotor mechanism plays in concus-

193. Gama, J. P.: Traité des plaies de tête et de l'encéphalite, ed. 2, Paris, Crochard, 1835, p. 101.

194. Jakob, Alfons: Experimentelle Untersuchungen über die traumatischen Schädigungen des Zentralnervensystems (mit besonderer Berücksichtigung der Commotio Cerebri und Kommotionsneurose.), in Nissl, F., and Alzheimer, A.: Histologische und histopathologische Arbeiten über die Grosshirnrinde mit besonderer Berücksichtigung der pathologischen Anatomie der Geisteskrankheiten, Jena, Gustav Fischer, 1913, vol. 5, p. 182.

195. Quoted by Jakob.[194]

196. Pirogoff, N.: Grundzüge der allgemeinen Kriegschirurgie, Leipzig, F. C. W. Vogel, 1864, vol. 1, p. 74.

197. Schmaus, Hans: Beiträge zur pathologischen Anatomie der Rückenmarkserschütterung, Virchows Arch. f. path. Anat. **22**:326, 1890.

198. Hashiguchi, Masaki: Experimentelle Untersuchungen über den traumatischen Hydrocephalus, Arb. a. d. Neurol. Inst. a. d. Wien. Univ. **29**:109, 1927.

199. Schonbauer, L.: Klinisches und Experimentelles über stumpfe Schädeltraumen, Beitr. z. klin. Chir. **137**:611, 1926.

sion. Their insistence on the significance of abnormality of the vasomotor tonus of the cerebral vessels seems justified. Wortis' [200] recent experimental work, in which he noted that animals are more susceptible to convulsions after head injuries, confirms in another way the general conception that one of the outstanding features of the postconcussion state is abnormal, labile intracranial circulation. The recent finding by Penfield [201] of nerve fibers around the blood vessels below the pia adds significance to these physiologic experiments. Most of the results of the more recent studies support the theory that derangement of the cerebral circulation is responsible for many of the sequelae of head injury. In spite of the evident limitations to the experimental method (Ricker [183]), it has helped to clarify many of the problems involved.

Given a continuously irritable and inefficient cerebral circulation, it is conceivable how mental activity, physical effort, intoxication, sensory stimulation and perhaps even changes in the weather (Blum [202]) can give rise to complaints. Mosso,[203] a number of decades ago, noted vascular changes in the brain in mental activity. Lennox,[204] using a newer, refined technic, recently showed how intellectual activity can affect the cerebral circulation. In Fulton's [205] patient with an occipital angioma there was intensification of bruit with visual effort. Alexander,[206] in 1912, ingeniously proved the effect of stimulation of the optic nerve on the flow of blood through the brain. It seems, therefore, that functioning of various parts of the brain is accompanied by changes in circulation—a not unexpected situation as such a reaction is found in most of the organs of the body. Any defect in the nicety of the regu-

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200. Wortis, S. B.: Head Injury: Effects and Their Appraisal: I. Experimental Studies of Induced Convulsions and Ventricular Distortions in the Cat, Arch. Neurol. & Psychiat. **27**:776 (April) 1932.

201. Penfield, Wilder: Intracerebral Vascular Nerves, Arch. Neurol. & Psychiat. **27**:30 (Jan.) 1932.

202. Blum, K:. Ueber die Abhängigkeit psychischen und nervösen Störungen von atmosphärischen Einflüssen, Arch. f. Psychiat. **96**:171 (March) 1932.

203. Mosso, A.: Ueber den Kreislauf des Blutes im menschlichen Gehirn, in Ueber das Verhalten des Blutkreislaufes im Gehirn während der verstärkten Vorstellungsthätigkeit und bei Gemüths- und Sinneseindrücken, Leipzig, F. C. W. Vogel, 1881, chap. 4, p. 62.

204. Lennox, W. G.: The Cerebral Circulation: XV. The Effect of Mental Work, Arch. Neurol. & Psychiat. **26**:725 (Oct.) 1931.

205. Fulton, J. F.: Observations upon the Vascularity of the Human Occipital Lobe During Visual Acuity, Brain **51**:310, 1928.

206. Alexander, F. G.: Untersuchungen über den Blutgaswechsel des Gehirns, Biochem. Ztschr. **44**:127, 1912. Alexander, F. G., and Revesz, G.: Ueber den Einfluss optischer Reize auf den Gaswechsel des Gehirns, Biochem. Ztschr. **44**:95, 1912.

lation of this vasomotor mechanism will result in exaggerated, protracted and even insufficient circulatory response to the stimuli enumerated.

In 1877, Mitchell [207] became interested in the relation between changes in the weather and pain, and reported observations on a keen army captain with stump neuralgia. Hellpach [208] carefully summarized the available data on the responses of the total organism to climatic variations and changes in the weather. Exacerbation of headache with a change of weather is well known in cases of head injury and may be related to alterations of cerebral circulation. Experimental study of the relation of the cerebral circulation to variations in weather would yield data of interest, especially in view of the recent developments in the technic of study of the cerebral circulation.

Pathologic Studies.—Examinations of the brains of patients who died in the postconcussion state as a result of other causes are not common. The conclusions to be drawn from such material are subject to the usual limitations of the morphologic method. Pathologic anatomy on the whole has not given much help in the understanding of the sequelae of head injury. Many of the older contributions are worthless. One of Macpherson's [209] case reports reads like the description of a patient with dementia paralytica who happened to fall. Jakob [194] pointed out that the cases of Friedmann,[35] Sperling and Kronthal [210] and Koeppen,[38] all of whom reported vascular lesions, were probably syphilitic. The importance of complicating diseases in explaining reported changes in the ganglion cells must be noted. This is especially evident in Obersteiner's [20] famous case. His patient had decubitus ulcers and a genitourinary infection and lingered considerably before he died.

Besides the gross lesions giving rise to focal changes and due to extensive laceration of brain tissue, many finer tissue alterations have been described as possible explanations for the vaguer complaints of patients with head injury. Bright's [211] observation, in 1831, of pinpoint

207. Mitchell, S. W.: The Relations of Pain to Weather, Being a Study of the Natural History of a Case of Traumatic Neuralgia, Am. J. M. Sc. **73**:305 (April) 1877.

208. Hellpach, W.: Die geopsychischen Erscheinungen, Wetter und Klima, Boden und Landschaft in ihrem Einfluss auf das Seelenleben, ed. 3, Leipzig, Wilhelm Engelmann, 1923.

209. Macpherson, John: Vacuolation of Nerve Cell Nuclei in the Cortex in 2 Cases of Cerebral Concussion, Lancet **1**:1127, 1892.

210. Sperling and Kronthal: Eine traumatische Neurose mit Sektionsbefund, Neurol. Centralbl. **8**:325, 1889.

211. Bright, Richard: Reports of Medical Cases Selected with a View of Illustrating the Symptoms and Cure of Disease by a Reference to Morbid Anatomy, London, Longman [and others], 1831, vol. 2, p. 403.

hemorrhages scattered throughout the brain has been corroborated by many authors (Rokitansky,[212] Blandin,[213] Foerster[214]). This same change was recently brought to attention again by Martland and Beling.[215] Jakob[194] denied that such hemorrhages can explain the clinical phenomena. Balado and Malbran[216] demonstrated that they can cause compression and distortion of ganglion cells. The changes in the ganglion cells first reported by Virchow[217] and later investigated by Budinger[218] and others need further study. Rossi recently pointed out the probability that a blow has a direct effect on protoplasm. The so-called microcellular structural changes described by von Sarbó are hypothetic alterations to account for reversible perverted function. Genewein[219] showed how the force of a blow on the head may be transmitted in a straight line directly to the ganglion cells. Vascular changes in the smaller vessels, said to occur only after trauma to the nervous system, have been reported by Ferraro,[220] Marburg[221] and Staemmler[188] (necrosis of the walls). Stevenson's[187] demonstration of continued microglial activity and Russel's[179] report of glial proliferation after head trauma may prove to be valuable observations.

More studies of the brains of patients long considered to be suffering from traumatic neuroses should be made with the newer histopathologic methods. Haase[222] has reported two such cases following head injury which were considered to be instances of traumatic neuroses and

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212. Rokitansky, C.: Handbuch der speciellen pathologischen Anatomie, Vienna, Braumüller und Seidel, 1844, vol. 2, p. 727.

213. Blandin, M.: Histoire de l'une des victimes de l'évenement du 8 mai, suivie de considérations sur la commotion cérébrale et sur les altérations viscérales qui resultent des grandes brûlures, Gâz. d. hôp. **4**:301 (June 2) 1842.

214. Foerster: Gehirnveränderungen bei Gehirnerschütterungen, Neurol. Centralbl. **23**:1063, 1904.

215. Martland, H. S., and Beling, C. C.: Traumatic Cerebral Hemorrhage, Arch. Neurol. & Psychiat. **22**:1001, 1929.

216. Balado, Manuel, and Malbran, Jorge: Clasificacion de las hemorrhagias post-traumaticas del cerebro, Arch. argent. de neurol. **7**:115 (Oct.) 1932.

217. Virchow, R.: Verkalkung abgestorbenen Gehirnzellen, Virchows Arch. f. path. Anat. **50**:304, 1870.

218. Budinger, K.: Ein Beitrag zur Lehre von der Gehirnerschütterung, Deutsche Ztschr. f. Chir. **41**:433, 1895.

219. Genewein, F.: Die mechanischen Vorgänge bei der Gehirnerschütterung und der Gehirncontusion, Beitr. z. klin. Chir. **128**:348, 1923.

220. Ferraro, A.: Contributio alla conoscenza dell'anatomia patologica della distruzione primitiva del midollo spinale (cosi detta necrose traumatica), Cervello **1**:361, 1922.

221. Marburg, Otto: Zur Pathologie der Kriegsbeschädigungen des Rückenmarks, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **22**:498, 1917-1919.

222. Haase, E.: Bemerkenswerte pathologisch-anatomische Befunde nach Gehirnerschütterung, Zentralbl. f. d. ges. Neurol. u. Psychiat. **54**:637, 1929-1930.

in which the patients died of other causes. He found rather extensive changes in the brain tissue, including softenings and glial proliferations. He advised caution in making a diagnosis of psychogenesis in cases of trauma to the head. Many more studies of such brains are needed, especially long after the initial injury. One must, however, look to physiology for a more satisfying explanation of disturbance of function.

The contemplation of the lacerations, pinpoint to larger hemorrhages and ganglion cell changes and of the numerous other demonstrable defects of nerve tissue in fatal cases makes one wonder as to the degree of cerebral damage in cases in which the patients return to consciousness and survive. One cannot help feeling that considerable diffuse damage to tissue must be present and that its presence accounts in part for the persistence of symptoms.

As far back as 1895, Strümpell [28] said: "That there are changes which may even be microscopic is shown by newer investigations, and it is not improbable that in an analogous way even commotio spinalis may lead to changes of a chronic nature."

CONCUSSION AND UNCONSCIOUSNESS

> *" . . . that a soldier in my presence gave to one of his fellows a stroke with a halberd on the head, penetrating even to the left ventricle of the brain, without falling to the ground." (Ambroise Paré [223])*

The complaints of injured persons without a clear history of unconsciousness are often listened to with a certain degree of skepticism and mistrust. A psychogenic syndrome is assumed to exist with minimal head injury. There is a widespread belief that a trauma severe enough to cause significant intracranial changes must result in unconsciousness. The possibility of concussion without unconsciousness is denied. This conception is implied in the definitions of writers on this subject. Groves,[143] for example, said that he had eight patients with residual defects of the eighth nerve following head injuries without concussion, that is, without loss of consciousness. Horn [40] related eight cases of convexity fracture without loss of consciousness and was somewhat hesitant about calling them instances of commotio cerebri. Bennet,[224]

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223. Paré, Ambroise: The Voyage of Perpignan, in Selections from the Works of Ambroise Paré, translated by Thomas Johnson, 1643, with a short biography and explanatory and bibliographical notes by Dorothea Waley Singer, London, John Bale Sons & Danielson, 1924, p. 173.

224. Bennet, William: Some Milder Forms of Concussion of the Brain, in Allbutt, T. C., and Rolleston, H. D.: A System of Medicine by Many Writers, London, The Macmillan Company, 1910, vol. 8, p. 231.

however, in Albutt's "System of Medicine," included in his classification of the milder forms of concussion of the brain a subgroup 4, "in which no loss of consciousness occurs at all."

We prefer to think of concussion clinically as a series of events resulting from a blow to the head severe enough to cause a disruption of intracranial equilibrium. If the blow happens to affect those parts of the brain concerned with the maintenance of the waking state, unconsciousness will result.

Neural units subserving the maintenance of the conscious state, wherever they may be, may be jarred only enough to give rise to an almost imperceptible obnubilation of consciousness, or they may not be affected. The effect of the blow may sometimes be limited to one part of the brain. Mossier [225] recorded an interesting instance in which the only result of a blow to the temporal region was an intense olfactory hallucinatory experience, without any loss of consciousness. The alteration in consciousness is frequently limited to what is known as the dazed state—temporary disorientation, without dissolution of erectness. It has been observed, for instance, that injuries to the occipital part of the head result in unconsciousness more often than do blows to the more anterior parts (Schück,[169] Laussmann [226]). There may therefore be no falling or staggering, and unless the injured person is watched carefully no change in behavior will be recorded.

It is strange how readily, on the other hand, the idea is accepted that rupture of relatively large intracranial vessels may take place without any loss of consciousness. This is true of subdural hematoma, epidural hemorrhage and even intracranial bleeding following head injuries. There are a number of reported, and many more unrecorded, cases of subdural hematoma in which trauma to the head was very mild, without even the history of a dazed state (Neuberger,[180] Thomas and his associates [227]). We recently observed a similar case.

CASE 14.—J. S., a high school boy, aged 17, three months before admission to the Mount Sinai Hospital on April 4, 1932, struck his head against a cross-beam while attempting to raise himself from a sitting position. He said that "things suddenly turned black for a moment," and except for transitory pains in the head for about two or three minutes he was entirely well until three days later, when he suddenly vomited. For the next three days he had severe headache and vomited

225. Mossier, G. de: Quelques symptômes rares consécutifs aux traumatismes crâniens, Rev. d'oto-neuro-opht. **10**:682, 1932.

226. Laussmann: Die Schädelverletzungen dreier Jahre im Urban Krankenhaus, Inaug. Dissert., Berlin, 1928.

227. Thomas, André; Schaeffer, H.; de Martel, T., and Guillaume, J.: Hématome sous- dural traumatique. Intervention opératoire. Guérison, Rev. neurol. **1**:94 (Jan.) 1932.

twice. For the following three months he was perfectly well, going to school, playing ball and moving around freely. Three days before admission the vomiting and headaches recurred.

On admission, there were: tenderness in the right parietal region; a positive Babinski sign on the left; bilateral papilledema, and xanthochromic spinal fluid. A craniotomy was done and an extensive chronic subdural hematoma was removed from the right side.

Another similar case was that of a motorman on a subway train who, following a mild injury to the head without loss of consciousness, died of a deep intracerebral hemorrhage.

CASE 15.—K. H., a man, aged 50, twenty days before admission to the Mount Sinai Hospital, while driving a subway train, struck his head against a heavy door. He did not lose consciousness at the time of the accident. He did not even relinquish his hold on the lever, which would have stopped the train immediately. He continued to run the train to the last station. The next morning he returned to work, but at that time he complained of headache, weakness of the left leg and some dizziness. The weakness on the left side became progressive, and twenty days after the accident he was admitted to the Mount Sinai Hospital. Exploration revealed nothing abnormal. Postmortem examination showed an intracerebral hemorrhage deep in the right parietal lobe. There was no evidence of a hemorrhage into a neoplasm, and no definite cerebral arteriosclerosis was noted.

In this case, without any clouding and without even momentary dazing, there was an injury severe enough to cause fatal bleeding from an intracerebral vessel. A more convincing case is that reported by Koopman.[228] A girl, aged 11 years, with no vascular or cardiac disease died after being struck on the head by a snowball without any loss of consciousness. The cause of death was a deep intracerebral hemorrhage. Bollinger,[229] Smidt,[230] Mendel [231] and others have also reported cases of late traumatic apoplexy (*Spätapoplexie*) with definite injury to the head but without loss of consciousness, and in some instances without even a history of momentary dazing. In addition, Jackson [232] and Josephowitsch [233] reported cases of traumatic epilepsy following head injury without loss of consciousness.

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228. Koopman: Weiterer Beitrag zur Frage des Hirntraumas und seiner tödlichen Folgen, Monatschr. f. Unfallh. **31**:98 (May) 1924.

229. Bollinger, O.: Ueber traumatische Spätapoplexie. Ein Beitrag zur Lehre von der Hirnerschütterung, in Festschrift Rudolf Virchow zu seinem 71 Geburtstage, Berlin, G. Reimer, 1891, vol. 2, p. 459.

230. Smidt: Zur traumatischen Spätapoplexie, Arch. f. klin. Chir. **142**:475, 1926.

231. Mendel, Kurt: Traumatische Spätapoplexie, Med. Klin. **25**:628, 1929.

232. Jackson, Hughlings: A Study of Convulsions, St. Andrew's M. Grad. A. Tr. **3**:167, 1870.

233. Josephowitsch, A. B.: Ueber die viscerale epileptische Aura (Zur Frage der kortikalen Lokalisation des Durstgefühls), Deutsche Ztschr. f. Nervenh. **123**: 43 (Nov.) 1931.

Ruhe [234] described a case of meningitis serosa diffusa following a slight head injury without loss of consciousness. Pitterlein [235] related a similar case with signs of a circumscribed serous meningeal reaction. Numerous reports of similar cases are available. We record here two cases of this type. In both the blow was severe enough to cause definite temporary disorder of the cerebrospinal mechanism immediately after the injury. In neither case was there any loss of consciousness.

CASE 16.—B. B., a man, aged 39, struck the left side of his head against an iron girder. He was not unconscious. He continued to work, with no particular complaints. The next day he complained of frontal headaches. The pains in the head increased in severity and became more constant. He had been well before the accident.

He was admitted to the Mount Sinai Hospital on June 13, 1926. Neurologic examination showed a meningeal syndrome (stiff neck and a bilateral Kernig sign), a suspicious Babinski sign, first on the right and later on the left, and bilateral papilledema. The spinal fluid pressure was over 560 mm. of water. After removal of 45 cc. of fluid, the pressure fell to 80 mm. There were 3 cells per cubic millimeter. The roentgenogram of the skull was normal. After the spinal puncture on June 15, the patient showed ataxia in the finger-to-nose test on the left side and some astereognosis in the left hand. He improved with repeated spinal punctures, but left against advice before the papilledema subsided. The Wassermann reactions of the blood and spinal fluid were negative.

CASE 17.—J. L., a policeman, aged 32, was admitted to the Morrisania Hospital on Jan. 20, 1933, complaining of headaches, dizziness and general weakness since an injury to the head six days before. He struck his head against the top of the doorway of his automobile as he was leaving it. There was no history of unconsciousness. He did not report any type of dazing. He continued on his way home, immediately noting a headache. He vomited once on the day after the accident.

On examination no scalp wound was apparent; the left pupil was a trifle larger than the right; there was slight deviation of the tongue to the right, and bilateral papilledema was present. The spinal fluid was clear and under a pressure of 28 mm. of mercury. There were no cells in the spinal fluid. The Wassermann reactions of the blood and spinal fluid were negative. The patient recovered completely with repeated spinal drainage and the intravenous administration of dextrose.

Knauer [67] stated that there may be absence of unconsciousness and vomiting in a true case of concussion, his conclusions being based partly on the behavior of animals after being struck on the head with a hammer. He noted excitement instead of stupor. Misdorf [236] suggested that occasionally mild injuries without loss of consciousness may injure defective, hypoplastic blood vessels.

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234. Ruhe, H.: Ueber die nosologische Stellung und Differentialdiagnose der sogenannten Meningitis serosa, Arch. f. Psychiat. **67**:459, 1922-1923.

235. Pitterlein: Zirkumskripte meningitis serosa, Deutsche med. Wchnschr. **35**:1772 (Oct. 7) 1909.

236. Misdorf, H.: Pathologisch-anatomischer und unfallrechtlicher Beitrag zur traumatischen Spätapoplexie, Aerztl. Sachverst.-Ztg. **38**:267 (Oct. 15) 1932.

It is probably within the experience of every one that sometimes after a blow to the head one feels dazed or confused for a minute or two. This transitory state cannot be explained on any basis other than that some disturbance in intracranial equilibrium took place—in other words, concussion. Prize-fighters have frequently been observed to fight after a severe blow and to have no recollection of it, their activity going on purely automatically. Tunney [237] described symptoms of mental dysfunction following a blow to the head which caused no unconsciousness:

> . . . One day while boxing with a sparring partner, Frank Muskie, we bumped heads. The part of my skull which is the thinnest, near the temple, struck the toughest part of his, the top. I was terribly dazed. As I straightened up a long, hard right swing landed on my jaw. Without going down or staggering I lost all consciousness of what I was doing and instinctively proceeded to knock Muskie out. Another sparring partner, Eddie Eagen, entered the ring; we boxed three rounds. I have no recollection of this, nor have I any recollection of anything that occurred until the next morning when I was awakened in my little cabin by the water's edge, wondering who I was and what I was doing there.
>
> As I lay in this awful state of returning consciousness I became greatly frightened. Gradually my name came to me. That I was a pugilist soon followed, then the thought of being champion—impossible—unbelievable. I must have had a long dream. Gradually came the realization that I had not been dreaming. I rose and asked guarded questions. I wanted to know all about the events of the day before. For 3 days I could not remember the names of my most intimate acquaintances. I had to stop training. I did not leave my cabin except to eat or take a short walk. On these occasions all seemed queer. I was unable to orient myself. The sensation I had was as though hot water had been poured through a hole in my skull and flowed down over the brain to my eyes, leaving a hot film. There were three newspaper men at camp reporting my activities. They had to be deceived. This story was too sensational to permit it to get out.
>
> I confided my condition to no one but Eddie Eagen. He was keeping a diary. It is amusing to read his notes of those few goofy days.
>
> After returning to normal, I decided that any sport in which such accidents could occur was dangerous. I realized I had a concussion. The first seed of retirement was sown then. The possibility of becoming punch drunk haunted me for weeks.

THE PSYCHOGENIC ELEMENT

It is necessary for once to abandon the specious dichotomy, organic and psychogenic, in an approach to cases of head injury. In most cases there is a subtle interaction of psychic and organic factors. A case is not either organic or functional. The result of the interaction of these biologic processes is not merely the sum of its parts. There is no clear juxtaposition, except in a few cases, of the psychologic and the organic. There emerges as a result of all this a clinical picture the interpretation of which requires at times considerable clinical acumen. It is the duty

237. Tunney, Eugene: A Man Must Fight, Collier's, March 26, 1932.

of clinicians to separate the primary organic effects of the injury to the head and the secondary psychogenic elaborations. Many have reiterated this plea.

The clinical features—headache, dizziness, irascibility, abnormal reaction to effort, vasomotor instability, fatigability, intolerance to intoxicants and to changes in the weather—as we have shown, are evidence in almost all cases of alteration of the activity of the intracranial tissues. These symptoms occur universally without conferences between patients who are widely separated from each other. Precisely the same reactions occur in nonlitigation patients as in those who can claim compensation. A similar symptom complex has been reported by Trautman [238] in electrical injuries. That the so-called secondary psychologic factors aggravate and prolong the reactions must be conceded, but they do not always create the difficulties. These persons are perforce required to adjust themselves to a difficult situation: inability to work, the necessity for the support of the family and the annoying subjective experiences. The mental reactions to chronic ailing and the socio-economic maladjustment usually come long after the original injury and must be clearly differentiated from the terror neurosis, a protracted psychologic reaction to the trauma situation, with the characteristic repetitive phenomena.

The suggestion has been made to limit the term "traumatic neurosis" to this anxiety reaction, which is present immediately following the accident and is apparently the result of the threat to ego-integrity. This response may occur in cases of mild head injury with little or no disturbance of the intracranial contents. The differential diagnosis of this anxiety syndrome from cerebral concussion must rest on clinical grounds in such cases. Restriction of this term to instances in which there has been no injury to the head is not clear. A mild concussion with no loss of consciousness may result in both an anxiety reaction and a postconcussion syndrome. We recently encountered an instructive case illustrating the possibility of the coexistence of the two syndromes. A man, aged 55, was in an automobile accident and had a mild injury to the head but no definite loss of consciousness. For about six weeks he was anxious, restless and sleepless and had repetitive dreams, reliving the trauma situation. Following the episode he apparently was left with a series of complaints characteristic of what we have called the postconcussion syndrome.

There can be no denying that the present mode of handling these unfortunate persons in compensation bureaus multiplies the psychic stresses and strains and complicates an already almost intolerable situ-

238. Trautman, E.: Ein Telephon-Unfall mit organischen Folgeerscheinungen in Gehirn und Rückenmark und seine Bedeutung für die Unfallbegutachtung, Deutsche Ztschr. f. Nervenh. **97**:63, 1927.

ation of life. The harshness, injustice and brutal disregard of complaints shown by the physicians and representatives of insurance companies and their ready assumption of intent to swindle do not foster wholesome patterns of reaction in injured persons. The frequent expression of unjustifiable skepticism on the part of examiners engenders resentment, discouragement and hopelessness and too often forces these people to resort to more primitive modes of response (hysterical). The repeated psychic traumas bring out the worst that there is in them and makes manifest all their frailties and constitutional insufficiences (Wagner [239]). This is especially true in view of the fact that the blow itself is known to give rise to defects in personality integration. In addition, the premorbid make-up of the injured persons varies considerably and undoubtedly contributes much to the manner in which they handle their problems. The trauma, moreover, lowers resistances and thresholds and brings prominently into consciousness repressed conflicts and difficulties.

A great deal has been said about the tendency of these patients to exaggerate and magnify their complaints. In addition to the gross and conscious type of exaggeration of the simulator, one must admit a natural tendency for these persons to exaggerate their complaints. The difficulties dominate the conscious field, and constant preoccupation with this type of mental content and introspection lead naturally to overevaluation and magnification. This reaction is also perhaps an effort to seize on the complaints in the occasionally frantic attempt of a patient to effect some sort of an adjustment. The psychogenic components of the whole picture are, from this standpoint, biologic attempts at adaptation, to a large extent subconscious. The hope for compensation often frustrates the possibility of the patient's disregarding or adjusting himself to annoying subjective experiences. There is no incentive to ignore discomfort and to keep working in spite of suffering. The identical series of complaints in injured athletes and in persons not entitled to compensation do not result, therefore, in as marked incapacity (case 13).

While we admit the frequent existence of a volitional trend, its rôle and significance can be evaluated only in the setting of the subconscious and organic factors to which we have already alluded. Its importance must not be overestimated. When such a volitional element is discerned, there exists an all too frequent tendency to consider the whole reaction as malingering and to evaluate the entire case from this angle.

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239. Wagner, Maria: Die Erbanlage bei Rentenneurotikern, Deutsche Ztschr. f. Nervenh. **123**:230 (Jan. 21) 1932.

The injured man with hemiplegia, blindness or an amputated limb finds that those about him understand his difficulties and appreciate his insufficiencies. He meets with sympathy and finds all making an effort to help him find his place in the community again. The injured man, however, with no focal signs or obvious defects but with only subjective complaints and a general lowering of his niveau of performance is met with coldness, suspicion and lack of sympathy. His complaints are not bulwarked by impressive handicaps or by convincing somatic distortions. This lack of recognition by those about him, together with the impending economic disaster, creates a serious situation which almost always results, if it lasts long enough, in the neurotic elaborations and symptom formations so often seen in these cases.

Many have protested against the rather vicious tendency to classify the neuroses following trauma with the behavior of swindlers and simulators. This practice contributes to the overlooking of organic features, around which the whole reaction sometimes develops. It must be reemphasized that the true neurosis is not a form of voluntary deception, though after it appears volitional factors may play a rôle in protracting the reaction.

Aschaffenberg,[56] among others, noted what little real benefit some of the injured persons derive from the compensation they receive when the amount is compared with their usual earning capacity. Many of these men repeatedly attempt to return to work but fail. There are undoubtedly other psychogenic factors besides the crass outlook for compensation to account for such somewhat paradoxical reactions, sometimes devastating to a man's career. There may be subconscious determinants for this behavior. We have already referred to this point of view. We recently observed the following instructive case.

Case 18.—D. S., an expert pastry baker, aged 34, was injured on May 26, 1932. While carrying a bag of flour on his back to a dough-mixing machine, he slipped and fell backward. He twisted his right foot and fell to the floor, striking the back of his head. He lost consciousness for a few moments. Immediately after the accident he complained of severe pain in the right foot. A fracture of one of the smaller bones in the right ankle region was confirmed by roentgen examination. The foot was immobilized in a cast for four weeks. Weakness and pain in the right foot persisted until the time of the first examination (Oct. 14, 1932) in spite of the fact that the fracture had healed. The patient also complained of headaches, especially on physical effort, occasional attacks of dizziness and some difficulty in thinking since the accident. He had made a number of attempts to return to work but had to go home because of headache and dizziness on movement as well as because of the persistent pain and weakness in the right foot.

Examination in October, 1932, revealed no focal signs of involvement of the central or the peripheral nervous system. There was a psychogenic right hemisensory syndrome, involving all the special senses on that side. There was marked weakness of all the muscles of the right foot and leg, which was most marked in

the toes. There was also some weakness of the muscles of the right upper extremity. The reflexes were all present and equal. There was no plantar response on the right.

The patient had earned about $75 a week as a pastry expert. He was receiving $25 a week compensation, and after a while this was withdrawn. The prospects for his receiving full compensation ($25) were slim at the time of the examination. This man realized the drastic cut in his income as a result of his incapacity. He made definite attempts to return to work on our advice. His premorbid make-up was excellent. He had always done well in his work. A careful psychodynamic approach in this case was not possible.

The patient derived little economic benefit from his continued incapacity. Perhaps a psychoanalytic approach would have revealed less evident, noneconomic reasons for his clinging to the illness. A closer study of this case and of similar ones has made us ask ourselves whether willingness to continue at an inferior level of adaptation is in itself a symptom of brain injury. This frequent peculiarly changed attitude toward reality, the satisfaction with a mere pittance and the occasional distinct regression to a lower level of existence suggest that the condition may be a sequel of a pathologic process in a few of the cases.

We have referred throughout this paper to the frequency of the coexistence of psychogenic and organic features in the same case. Charcot [240] and Babinski [241] long ago emphasized the frequency with which organic and functional disease may be found in the same patient. In nine of nineteen patients studied there were undoubted psychogenic features in addition to evidence of organic involvement of the nervous system. Such psychogenic superimposition is much more common than is usually admitted and is more likely to occur the longer the injured person is permitted to go along without some satisfactory solution of his difficulties. We found definite psychogenic complications immediately after the trauma in only one of the nineteen cases studied, and in this case there was a history of hysterical conversion phenomena before the accident (case 13).

The generalization that hysterical conversion mechanisms appear only when there are no physical sequelae or only mild somatic residuals is not entirely borne out by experience. Voss and Meyer [242] attempted to explode this theory on the basis of their experiences during the war. Goodhart and Savitsky [243] recently showed that psychogenic superim-

240. Charcot, quoted by Babinski.[241]

241. Babinski, J.: Association de l'hystérie avec les maladies organiques du système nerveux, les névroses et diverses autres affections, Bull. et mém. Soc. méd. d. hôp. de Paris **9**:775, 1892.

242. Voss, G., and Meyer, G.: Zur Begutachtung der Schädelverletzungen, Nervenarzt **3**:129 (March 15) 1930.

243. Goodhart, S. P., and Savitsky, N.: Neuropsychiatric Disorders Simulating Primary Medical and Surgical Disorders, M. J. & Rec. **135**:331 (April 6) 1932.

position may occur even with serious somatic disease. While it is true that usually these functional additions appear in cases of lesser severity, one cannot reason that because such psychogenic phenomena exist there can be no serious injury and only minor, negligible defects. Each case must be judged on its merits. One must be cautious in concluding from the presence of a few psychogenic features that the whole syndrome is psychologic. We have already referred to this problem in the discussion of the patient with alexia (case 4). We were much surprised in the following case by the encephalographic findings, which pointed to the probable existence of extensive intracranial changes.

Case 19.—J. M., a Russian Jewish tailor, aged 59, was admitted to the Mount Sinai Hospital complaining of heart burn of four years' duration. The sensation frequently ascended along the sternum to the mouth. He was not relieved by sodium bicarbonate. For about a year he had been unsteady on his feet. The gait had become staggering. He would fall toward the right side and dragged the right lower limb. For a year he had had severe attacks of dizziness. Deafness had been increasing for four years. Forty years before admission he sustained a severe injury to the right side of the head and was unconscious for a short time.

Examination revealed no objective evidence of focal injury to the nervous system, except bilateral nerve deafness, which was undoubtedly intensified by psychologic factors. In spite of complaints of weakness, no evidence of focal disease was found. There were frequent bizarre movements of the limbs and body, especially when he was disturbed emotionally. The gait was peculiar. There seemed to be a discrepancy between his behavior, his complaints and the objective findings. Vestibular tests gave normal results. There was bilateral myopia of high degree.

Encephalography was done on April 5, 1932. After the removal of over 200 cc. of fluid and the injection of 160 cc. of air he continued to remain in good condition. There was moderate symmetrical internal hydrocephalus. There were marked enlargement of the subarachnoid space and marked retraction of the brain on the right side, with a rather broad adhesion in the postparietal area.

Most of those who observed this man thought that the whole reaction was probably psychogenic and that there was no relation between the present condition and the old injury. Encephalography proved beyond a doubt the existence of an organic change in the brain, possibly the result of the old head injury. The many years of freedom from subjective complaints makes one rather hesitant about connecting the present condition with the trauma many years before. It may be that something had happened in the past few years to disturb the equilibrium within the skull, which had been adjusted many years before.

We have already commented on the absence of subjective complaints in case 7. It is evident that in spite of extensive intracranial defects and destruction of neural tissue a sort of equilibrium may be reached after a while. This freedom from symptoms may persist, and most probably involves the continued operation of compensatory mechanisms. In

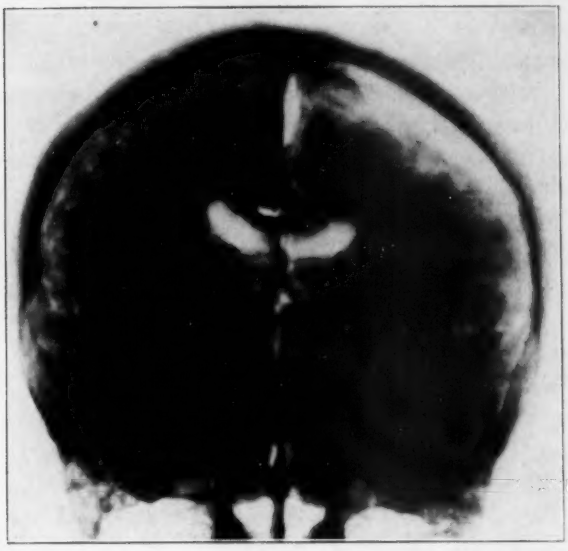

Fig. 9 (case 19).—Encephalogram showing moderate internal hydrocephalus and an increased amount of air over the left hemisphere.

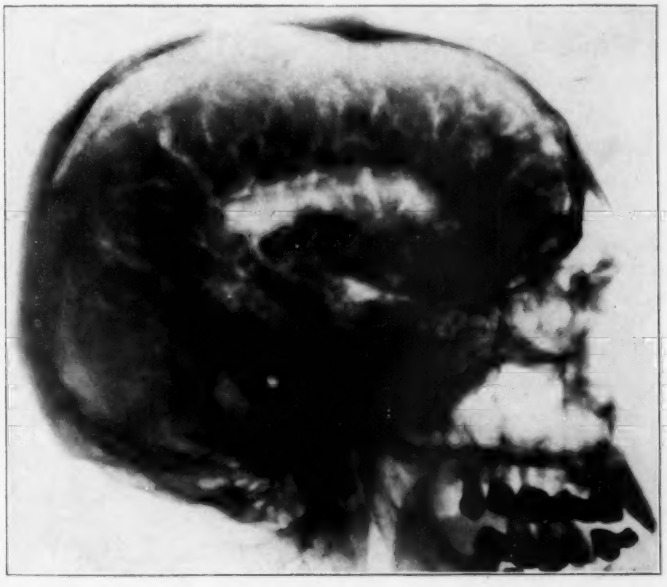

Fig. 10 (case 19).—Encephalogram showing internal hydrocephalus and marked increase of air over the cerebral hemispheres.

neither case 7 nor case 19 was there any question of reopening of litigation.

The important problem is the reduction to a minimum of the secondary psychologic reactions, which after a time dominate the entire clinical picture. The cooperative investigation suggested by us will have a salutary effect on the injured person. It will discourage simulation and will do much to encourage and comfort him. We have not observed that these examinations result in further psychogenic complications. The whole procedure gives the patient the impression that he is being dealt with fairly and that his complaints are being received seriously.

CONCLUSIONS

1. We have limited our consideration to cases of actual physical injury to the head.

2. Clinically, the organic sequelae of head injury should be differentiated from the terror and anxiety reactions following a threat to bodily integrity. The term "traumatic neurosis" may be confined to the latter type of reaction (Fenichel,[244] Kardiner [245]).

3. In our opinion, the subjective posttraumatic syndrome, characterized by headache, dizziness, inordinate fatigue on effort, intolerance to intoxicants and vasomotor instability, is organic and is dependent on a disturbance in intracranial equilibrium due directly to the blow on the head. We suggest the term "postconcussion syndrome" for this symptom complex.

4. "Traumatic encephalopathy" may be used as a generic term if it is understood that it includes cases in which physiologic disturbances of the cerebral mechanism are present although organic lesions are not demonstrable.

5. Psychogenic factors always complicate the clinical picture if the socio-economic and other difficulties following the trauma last long enough.

6. Negative results on neurologic examination and a normal mental status are no final criterion of the presence, absence or degree of damage to the brain resulting from head trauma.

7. No opinion should be formed in doubtful cases without a systematic clinical survey, such as is suggested by our cooperative investigation.

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244. Fenichel, Otto: Outline of Clinical Psychoanalysis, Traumatic Neurosis, Psychoanalyt. Quart. **1**:572, 1932.

245. Kardiner, Abraham: The Bio-Analysis of the Epileptic Reaction, Psychoanalyt. Quart. **1**:375, 1932.

8. Observations should be recorded regardless of the examiner's opinion regarding their significance at the time of the examination.

9. Failure to understand a clinical phenomenon is no proof of its psychogenicity.

10. Significant intracranial injury can occur without loss of consciousness.

11. More attention should be paid by neuropsychiatrists to the methods of investigation developed by experimental and clinical psychologists in the study of head injuries and their sequelae.

BRAIN TRAUMA

HISTOPATHOLOGY DURING THE EARLY STAGES

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Statistics show an appalling incidence of head trauma. The subject of the changes in the brain and the symptoms resulting from head injuries is coming to be most important in modern medicine. The courts are deluged with cases in which compensation and redress are sought because of claims of permanent sequelae as the result of alleged injuries to the brain. The subject is further complicated by the fact that neurologists and neurosurgeons are still at odds concerning the question of the organic or functional nature of many of the symptoms.

The clinical evidences of brain trauma during the acute period require no lengthy descriptions. It is well known that the duration of unconsciousness is usually in direct proportion to the severity of the brain injury, although this does not always prove an accurate estimate, since complications, such as hemorrhage, can increase the duration of loss of consciousness. General shock is the rule. Evidences of gross damage to the brain may be present. Fracture of the skull is not a necessary accompaniment of severe damage. Paralysis and other focal evidences are usually lacking, although as the result of gross bleeding there may be focal symptoms.

The symptoms occurring after head trauma are usually so uniform, even in the absence of legal complications, that every writer has called attention to this remarkable fact. Dana,[1] in addressing the American Neurological Association, enumerated the symptoms as follows: headache, vertigo, insomnia, irritability, anxiety, depression, memory defects, liability to fatigue, tinnitus, partial deafness and loss of weight. Evidences of focal brain damage are usually in the background. While Dercum and Sachs were in sympathy with Dana's conclusion that the symptoms were probably the result of desire for compensation,

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From Temple University School of Medicine, Philadelphia.

Read at the Fifty-Ninth Annual Meeting of the American Neurological Association at Washington, D. C., May 9, 1933.

1. Dana, C. L.: Wounds of the Head and Compensation Laws, Arch. Neurol. & Psychiat. **4**:479 (Nov.) 1920.

Cushing stood out for the idea that in severe brain trauma serious damage occurred to the central nervous system and caused people to become crippled just as severely as if they had lost their legs. He acknowledged, of course, that a functional overlay might be present, but that in a large proportion of cases the victims of serious cerebral contusions were permanently handicapped.

It has, therefore, been of the utmost importance that methods for the study of the appearance of the brain during life be devised in an effort to determine the extent of damage. For this reason encephalographic study has rendered great service, to both patient and doctor, in disclosing the gross lesions of the brain as the result of trauma (Wartenberg,[2] Foerster,[3] Bielschowsky,[4] Fay,[5] Hauptmann[6] and Friedman[7]). One does not, of course, have any method for comparison of the conditions before and after injury, and the deductions are based on the assumption of a normal prior state.

The brain of a patient who dies hours after a severe head trauma often shows a subarachnoid hemorrhage, with maceration of the brain on the undersurface of the frontal and temporal lobes. This finding has been so uniform that an explanation for it has been sought, and we have concluded that it is the result of lack of an adequate water bed between these parts of the brain and the base of the skull. Gross hemorrhage in the brain substance is fairly common, with contusion or maceration of the tissue in the immediate neighborhood. The hemorrhage, far more frequently than is thought, is due to contrecoup. Subdural bleeding (Wertheimer,[8] Coleman[9] and Gardner[10]) is a less frequent finding, and epidural hemorrhage is comparatively rare. Late hemorrhages in these areas have been fully described in the literature

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2. Wartenberg, R.: Arch. f. Psychiat. **77**:507, 1926; Ztschr. f. d. ges. Neurol. u. Psychiat. **94**:585, 1925.

3. Foerster, O.: Ztschr. f. d. ges. Neurol. u. Psychiat. **94**:512, 1925.

4. Bielschowsky, P.: Störungen des Liquorsystems bei Schädeltraumen, Ztschr. f. d. ges. Neurol. u. Psychiat. **117**:55, 1928.

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6. Hauptmann, A.: Die Objektivierung postcommotioneller Beschwerden durch das Encephalogramm, Arch. f. Psychiat. **96**:84, 1932.

7. Friedman, E. D.: Head Injuries: Effects and Their Appraisal; III. Encephalographic Observations, Arch. Neurol. & Psychiat. **27**:791 (April) 1932.

8. Wertheimer, P.: Traumatic Subdural Hemorrhage in Adults, Rev. de chir., Paris **61**:150, 1932.

9. Coleman, C. C.: Intracranial Hemorrhage Following Head Injury, South. M. J. **21**:697 (Sept.) 1928.

10. Gardner, W. J.: Traumatic Subdural Hematoma with Particular Reference to the Latent Interval, Arch. Neurol. & Psychiat. **27**:847 (April) 1932.

(Goroncy,[11] Smidt[12] and Eck[13]). On section of the brain, aside from gross hemorrhages, contusions and macerations, one frequently finds intense congestion, edema and petechiae. The latter are frequently limited to the subcortical white matter, as in various infections, intoxications and blood dyscrasias. In cases in which life is prolonged, the damage to the brain is usually less severe.

From the histologic point of view, the changes that result from trauma have been investigated mostly from special angles. It is with the purpose of attempting to determine all the minute changes in the brain after trauma and to correlate the clinical and histologic features that this presentation is made.

From the historical angle, large series of cases of head trauma were not reported until the advent of the automobile. LeCount and Apfelbach[14] reported the first large series, 504 cases, all from the coroner's office in Chicago. They described the types of gross lesions found and stressed the frequency of lacerations on the outside of the brain. Their theory was that when the brain injury occurred when the head was in motion there was a likelihood of contrecoup lesions (182 cases), but when the injury occurred with the head at rest the lesion was usually beneath the point of impact.

Apfelbach,[15] in a series of cases, noted an absence of edema if the patient died within a few hours, or survived three or more days after the injury. He concluded that most of the intracerebral lesions were due to rupture of the vessels deep within the cortex.

Cassasa[16] described 5 cases with multiple punctate hemorrhages throughout the cortex after head injury, without laceration of the scalp, fracture of the skull or laceration of the cortex.

Trotter[17] claimed that concussion causes amnesia for the injury and he believed it to be the result of an actual organic injury. He was able

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11. Goroncy, C.: Traumatische Spätblutung im Gehirn, Deutsche med. Wchnschr. **49**:413 (March 30) 1923.

12. Smidt: Zur traumatischen Spätapoplexie, Arch. f. klin. Chir. **142**:475, 1926.

13. Eck, H.: Beitrag zur Lehre der traumatischen Spätapoplexie, Virchows Arch. f. path. Anat. **284**:67, 1932.

14. LeCount, E. R., and Apfelbach, C. W.: Pathologic Anatomy and Traumatic Fracture of Cranial Bones and Concomitant Brain Injuries, J. A. M. A. **74**:501 (Feb. 21) 1920.

15. Apfelbach, C. W.: Traumatic Fractures of Cranial Bones: I. Edema of the Brain, Arch. Surg. **4**:434 (March) 1922.

16. Cassasa, C. B.: Multiple Traumatic Cerebral Hemorrhages, Proc. New York Path. Soc. **24**:101, 1924.

17. Trotter, W.: Shell Wound of the Head, Brain **42**:353, 1919; On Certain Minor Injuries of the Brain, Brit. M. J. **1**:816 (May 10) 1924; Lancet **1**:935 (May 10) 1924.

to see, at operation, incomplete resolution of a contusion of the brain four years after the accident, and postulated that lesions heal very slowly. The headache that so often results was thought to be due to increased intracranial pressure and stretching of the meninges.

Osnato and Giliberti [18] believed that definite brain injury occurred as the result of concussion and often resembled an encephalitic process. They thought that the clinical sequelae were secondary to degenerations which occurred.

Martland and Beling,[19] in 309 cases in which gross studies alone were made, included traumas of all sorts. They found capillary foci of bleeding in the basal ganglia and by contrecoup. They believed that multiple concussion hemorrhages might explain many of the sequelae of cranial injuries. As a whole, similar conclusions have been given by McConnell,[20] Vance,[21] Symonds,[22] Pommé and Liégeois,[23] Rossi,[24] Ritter,[25] Spatz [26] and Russell.[27] Beekman [28] showed that children withstand head trauma better than adults and have fewer sequelae. Strecker and Ebaugh [29] gave an excellent summary of the sequelae of head trauma in children. Following the World War many good papers on

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18. Osnato, M., and Giliberti, V.: Postconcussion Neurosis—Traumatic Encephalitis: A Conception of Postconcussion Phenomena, Arch. Neurol. & Psychiat. **18**:181 (Aug.) 1927.

19. Martland, H. S., and Beling, C. C.: Traumatic Cerebral Hemorrhages, Arch. Neurol. & Psychiat. **22**:1001 (Nov.) 1929.

20. McConnell, A. M.: Sequelae of Minor Head Injuries, Irish J. M. Sc., Jan., 1926, p. 29.

21. Vance, B. M.: Pathologic Conditions Complicating Fractures of the Skull, Arch. Neurol. & Psychiat. **19**:959 (May) 1928.

22. Symonds, C. P.: Observations on Differential Diagnosis and Treatment of Cerebral States Consequent upon Head Injuries, Brit. M. J. **2**:829 (Nov. 16) 1928; The Effects of Injury upon the Brain, Lancet **1**:820 (April 16) 1932.

23. Pommé, B., and Liégeois, R.: Au suject du syndrome subjectif commun des blessés du crane, Rev. neurol. **1**:483 (April) 1930.

24. Rossi, A.: Linee fondamentali di traumatologia del sistema nervoso centrale Riv. di pat. nerv. **38**:797, 1931.

25. Ritter, A.: Concussion of the Brain, Deutsche Ztschr. f. Chir. **175**:10, 1922.

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27. Russell, W. R.: Cerebral Involvement in Head Injury, Brain **55**:549, 1932.

28. Beekman, F.: An Analysis of Three Hundred and Thirty-One Cases of Injury to the Head in Children with Especial Reference to End-Results, Arch. Neurol. & Psychiat. **19**:955 (May) 1928.

29. Strecker, E. A., and Ebaugh, F. G.: Neuropsychiatric Sequelae of Cerebral Trauma in Children, Arch. Neurol. & Psychiat. **12**:443 (Oct.) 1924.

war injuries were published (Lister and Holmes,[30] Cushing,[31] Head[32] and Shields[33]). Von Sarbó[34] recently summarized the microscopic changes in the brain found in cases of injury during the war.

From the microscopic angle, one of the earliest contributions was by Adolf Meyer,[35] who found as residuals small foci of softening or defects in the cortex, especially in the frontal and temporal lobes. There may be residuals of old bleeding in the foci of softening. There was absence of the marginal glia beneath the point of injury. In addition, the diffuse effects were glial alterations with many small foci or other organic degenerations throughout the brain in many cases. Somewhat similar findings were described prior to Meyer by Friedmann,[36] and also later by Wohlwill[37] and Jakob,[38] and clinically by Hadley.[39] Mott[40] explained actual "shell-shock" during the World War on the basis of multiple petechial hemorrhages throughout the brain.

Martland[41] thought that the symptoms which indicated degenerative changes in the brain of prize-fighters whom he called "punch drunk" were due to multiple capillary hemorrhages.

REPORT OF CASES

CASE 1.—*History.*—J. S., a white man, aged 29, was admitted to Temple University Hospital on May 6, 1932, and died on May 10. While working on a roof, he had tripped and fallen a distance of about 20 feet. He was brought to the hospital in a semistuporous condition.

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30. Lister and Holmes, Gordon: Proc. Roy. Soc. Med. (Sect. Ophth.) **9**:57, 1916.

31. Cushing, H.: Study of a Series of Wounds, Involving the Brain and Its Enveloping Structures, Brit. J. Surg. **5**:58 (April) 1918.

32. Head, H.: Shell Wound of Head, Brain **42**:349, 1919.

33. Shields, O.: Gunshot Wounds of Head, Brain **42**:355, 1919.

34. von Sarbó, A.: Die mikrostrukturellen traumatischen Veränderungen des Nervensystems im Lichte der Kriegserfahrungen, Schweiz. Arch. f. Neurol. u. Psychiat. **29**:127, 1932.

35. Meyer, A.: The Anatomical Facts and Clinical Varieties in Traumatic Insanity, Am. J. Insan. **60**:374, 1904.

36. Friedmann, M.: Ueber eine besondere schwere Form von Folgezuständen nach Gehirnerschütterung und ueber den vasomotorischen Symptomen complex bei derselben im Allgemeinen, Arch. f. Psychiat. **23**:230, 1891.

37. Wohlwill, F.: Zum Kapitel des posttraumatischen Psychosen, Monatschr. f. Unfallheilk. **20**:73, 1913.

38. Jakob, A.: Ueber einen besonderen Fall von Kommotionspsychose, Ztschr. f. d. ges. Neurol. u. Psychiat. **51**:30, 1919.

39. Hadley, E. E.: Mental Symptoms Following Complex Cranial Trauma, J. Nerv. & Ment. Dis. **56**:567, 1922.

40. Mott, F. W.: Microscopic Examination of the Brain in Death from "Shell Shock," Brit. M. J. **2**:612, 1917.

41. Martland, H. S.: Punch Drunk, J. A. M. A. **91**:1103 (Oct. 13) 1928.

Examination.—The patient was well developed and extremely restless, but could be aroused momentarily. There was a contused area over the left parietal region. No depression or crepitus was made out. Roentgen examination of the skull, made immediately after admission, showed a fracture of the left parietal bone. The pupils reacted normally. There were no cranial nerve palsies. The eyegrounds showed no abnormalities. There was bleeding from the left ear. The deep reflexes were present throughout. The abdominal reflexes were absent. There was no weakness of any of the limbs. The blood pressure was 135 systolic and 85 diastolic; the pulse rate, 110; the temperature, 99.60 F., and the respiratory rate, 35. Spinal puncture showed a pressure of 35 mm. of mercury; the fluid at first was clear, then pink, and finally bright red.

Course.—During the four days in the hospital the patient was markedly confused and restless. No new physical signs developed, and improvement seemed to be marked within the first twenty-four hours. In view of this he was treated conservatively, and spinal punctures were done infrequently. Two days after the injury, evidences of cerebral compression began, with beginning weakness of the right side of the body, and with Cheyne-Stokes respiration. Because of the suspicion that a focal lesion was present, exploration of the left temporal region was done under local anesthesia, and a large subdural hematoma was uncovered and removed. The brain appeared edematous. After the operation the patient did not rally; he died four days after the accident.

Pathologic Examination.—The heart showed early arteriosclerosis of the coronary vessels, with severe myocardial degeneration. The lungs showed terminal hypostatic pneumonia. The spleen was congested and enlarged. The kidneys revealed passive congestion, with numerous petechial hemorrhages. There was an old cholelithiasis as well as a hematoma of the diaphragm.

Gross examination of the brain showed a considerable amount of blood in the subarachnoid space. There was hemorrhage into the cerebral tissue in the left parietal area. Edema was not a feature.

Microscopic examination of the central nervous system showed varying amounts of fresh blood within the subarachnoid space. The blood vessels were all markedly congested. A mild fibroblastic proliferation was in progress. Occasionally one found, deep in the various fissures, a collection of phagocytic elements, most of which contained masses of blood pigment. Adhesions to the cortex could be made out in various places, with penetration of the new vasculature from the pia into the adjacent cortical tissue.

The cortex, as a whole, showed no disturbances in its architectural arrangement. The outstanding general features of the cortex consisted of a prominence of the small blood vessels and a general, comparatively uniform, gliosis. Scattered at irregular intervals throughout the brain substance one found small hemorrhages, all of which were practically of the same age (fig. 1). One large hemorrhage was present in the parietal lobe. While it was true that the vessels, in the neighborhood of the hemorrhages particularly, showed swelling of their lining cells as well as beginning vascularization, those in the rest of the cortex were by no means exempt from the endarteritic change. The large hemorrhage within the parietal lobe showed nothing unusual in its microscopic picture. There had been, as is usual with hemorrhages of this sort, complete destruction of the brain tissue in the immediate neighborhood of the hemorrhage, with minute petechiae in the surrounding tissue. There was a great deal of maceration of tissue immediately surrounding the hemorrhage, which merged into normal tissue more or less imperceptibly, giving a yellowish tinge to the brain substance in the immediate neighborhood.

There had been a rupture of the hemorrhage into the subarachnoid space. In some of the smaller areas surrounding the large hemorrhagic focus one occasionally found groups of polymorphonuclear cells, giving the impression at first glance of petechial abscesses.

Smaller hemorrhages were scattered irregularly throughout the entire brain. Here, too, there was no preference as far as location was concerned, since they were frequently in both cortex and subcortex in adjacent portions of the brain substance. These were miniatures of the large hemorrhage. One noted beginning vascularization even within the blood elements themselves, while immediately sur-

Fig. 1 (case 1).—Cortex, showing petechial hemorrhages with loss of normal structure, surrounding reactive gliosis and vascularization. Toluidine blue stain.

rounding the hemorrhagic foci proliferation of new blood vessels was going on very actively. There was also, as a general rule, proliferation of the glia immediately surrounding the hemorrhage, although the cortex showed such a uniform gliosis that the slight increase in these focal areas was not remarkable. While phagocytic elements were in the great minority, one frequently could find small collections in the neighborhood of the hemorrhage.

While it is stressed that a generalized gliosis of the entire brain was actively in progress, with the ordinary toluidine blue stain this process was concealed by the great numbers of ganglion cells that are ordinarily present within the cortex. At the marginal layer the gliosis stood out more vividly, and here one could see

a definite increase in the glia elements which in places reached such a marked degree that the demarcation between the first and the second cortical layers was indistinct.

As far as the small blood vessels were concerned, one found that, as a rule, they stood out distinctly, not so much because most of them were dilated, owing to the congestion which was present uniformly, as on account of the prominence of the lining elements. In the slightly larger blood vessels one found that many were beginning to show hyalinization of their walls, although as a rule this was by no means excessive.

Fig. 2 (case 1).—Cerebellum showing softening and vascularization in the molecular layer, with breaking up of the granular layer. Toluidine blue stain.

In the subcortex there was no great rarefaction of tissue. One did find areas in which the normal oligodendroglia had disappeared and had been replaced by astrocytes. Occasionally one found areas in which the blood vessels were surrounded by gitter cells, usually containing some blood pigment.

The cerebellum in this case merits more than passing mention. While clinically no disturbance in cerebellar function had been noted, microscopic study demonstrated that scattered throughout the entire cerebellar substance were many areas of softening at irregular intervals. These areas consisted of collections of gitter cells with beginning vascularization. The softened areas were not limited to any one anatomic area, but there was involvement of the molecular and granular

layers equally. In the molecular layer the areas of softening had occasioned a complete disappearance of the normal elements, which were replaced by a network of new blood vessels between which gitter cells were located (fig. 3). In the granular layer, however, the disappearance was rather remarkable. There had apparently been a scattering of the small granular cells even into the adjacent regions of the molecular layer; the granular cells themselves appeared to be markedly "thinned out," and the entire granular layer gave the impression of being "moth eaten" (fig. 2). In the areas in which softening had taken place there was a complete disappearance of the Purkinje elements. No increase in the Bergmann

Fig. 3 (case 1).—Higher magnification of figure 2, showing details of vascularization, accumulation of gitter cells and meningeal infiltration, as well as fragmentation of the granular layer.

glial layer had taken place. Frequently one saw smaller areas which seemed to have a relation to the blood vessels in the meninges (fig. 4), in which the meninges themselves contained markedly congested blood vessels with some blood free in the meshes and with numerous round cells immediately surrounding the congested blood vessels. The areas of necrosis seemed to radiate away from or toward these foci, and the gitter cell elements appeared to be making their way toward the subarachnoid space. Fat stains of areas of this sort showed that the majority of the cells were gitter elements, and while one occasionally had the impression that some of the deeply stained round nuclei, mostly within the subarachnoid space, were

lymphocytes, the deduction is clear that if lymphocytes were present at all within the cellular group they were in the minority and only brought out as the result of a secondary or symptomatic inflammation.

It must not be concluded from the description here given that no portion of the cerebellar hemisphere was normal, because whole stretches of cerebellar tissue were absolutely within normal limits.

The dentate nuclei merit special consideration. While many ganglion cells were still present, there was no doubt that there had been a considerable loss. This was evidenced not only by the decrease in the number that is usually seen within this

Fig. 4 (case 1).—Cerebellum showing generalized softening in the molecular layer with focal vascularization and migration of phagocytes into the meninges. Toluidine blue stain.

nucleus, but also by the great glial increase within the nucleus itself. Many of the smaller blood vessels within the nucleus, and around it, showed perivascular collections of phagocytic elements, many of which still contained some débris within the neighborhood of one of the dentate nuclei. Small hemorrhages could be made out, with considerable destruction of the tissue in the immediate neighborhood. This differed in no way from that seen within the cerebrum, either in appearance or in the general reaction of the tissue. The blood vessels within the cerebellum require no special description as they conform in every detail to those seen in the cerebrum.

Comment.—In this patient, who died four days after the injury, although there had been a great deal of hemorrhage as the result of the trauma in the form of a comparatively large hemorrhage, such as frequently occurs, there were minute hemorrhages scattered throughout the cerebrum and less widely in the cerebellum. One is not surprised at such a finding in the brain after a trauma of great severity.

However, the cerebellar picture here is of more interest. One of the outstanding complaints in the posttraumatic period is of dizziness, usually associated with some headache. While dizziness occurs frequently from cerebral lesions, it is probably true that this symptom might be greater in cases in which cerebellar damage had occurred. The impression of the picture as seen under the microscope is that of a commotio cerebelli rather than the result of vascular occlusion in many areas. There were many foci that bore a striking resemblance to areas from vascular occlusion. There was probably, therefore, a dual process here: (1) the result of maceration of tissue from the concussion at the time of the trauma, and (2), the necrosis as a result of obstruction to the blood supply from either swelling of tissue or the presence of so much free blood within the subarachnoid space. In a case of this sort, had not the large cerebral hemorrhage produced death one could easily understand that this patient would have had a great deal of dizziness for a long time. The presence of cerebellar signs, with nystagmus and incoordination, might have been present early in the condition, but as is well known destruction of cerebellar tissue frequently causes much less disturbance to its normal function than would an expanding lesion within its substance.

The pial cortical adhesions seen in this case are also important. There was no doubt from the histologic picture that had the patient recovered, many adhesions of the pia to the adjacent cortex would have been left. These may be the cause of the frequent headache which results as a posttraumatic symptom. It may explain also the occasional relief that one obtains in cases of this sort from the therapeutic injection of air (Penfield [42]). It may also throw light on the convulsive seizures that one observes occasionally as a posttraumatic symptom.

CASE 2.—*History.*—A white woman, aged 39, who had been working as a paper-box maker, was admitted to the accident ward, in the neurosurgical service of Dr. Fay, on June 17, 1931, in profound shock, with a depressed fracture of the skull in the left occipital region, laceration of the scalp, and fracture of the left humerus. According to the history, she had been struck on the right side of the body by an automobile and hurled a distance of approximately 35 feet, and was immediately rendered unconscious.

42. Penfield, W.: Meningocerebral Adhesions: A Histological Study of the Results of Cerebral Incision and Cranioplasty, Surg., Gynec. & Obst. **39**:803 (Dec.) 1924; Mechanism of Cicatricial Contraction in the Brain, Brain **50**:499, 1927.

Examination.—The patient was obese and in coma. There was a deep depressed fracture of the skull on the right side in the occipital region. The face was cyanotic. There was evidence of weakness of the left side of the face. The pupils were both contracted, but reaction to light was still present. There was a tendency for the eyes to be turned to the right when they were forced open. There was no trauma to the face. Blood was present in the mouth. Breathing was stertorous, and the rate was 24. The pulse rate was 96; it was regular but faint. The blood pressure was 108 systolic and 78 diastolic. The knee reflexes were not obtained. The left forearm was in a splint, and both legs showed subcutaneous hematomas.

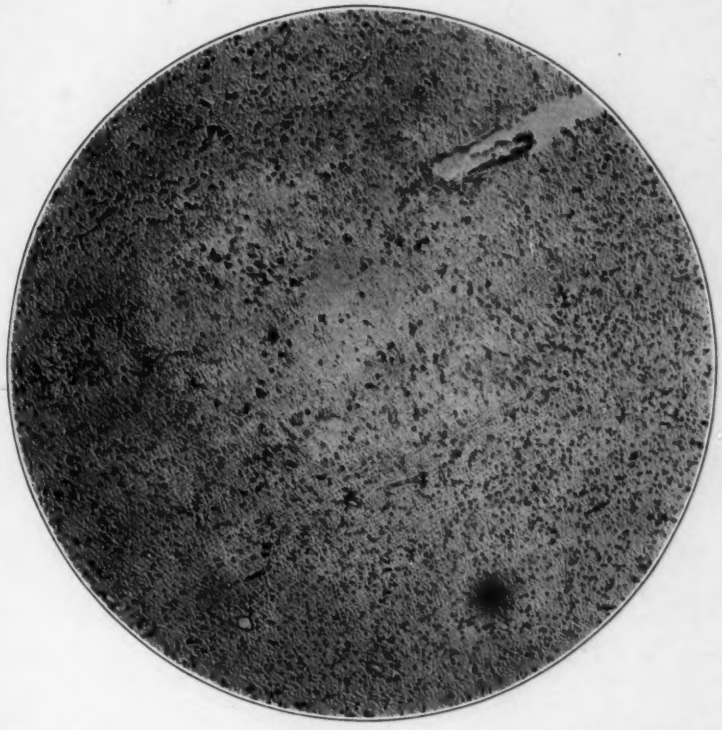

Fig. 5 (case 2).—Subcortex, showing petechial hemorrhage, with glial reaction around it. Toluidine blue stain.

Course.—Twenty-four hours after admission the patient could be aroused sufficiently to give her name and address. She was still cyanotic, and breathing was difficult. Spinal puncture showed an initial pressure of 12 mm. of mercury. The first 5 cc. removed was clear and colorless; afterward the fluid contained a great deal of blood. The patient was treated for shock. Within twenty-four hours a Babinski sign was elicited on the left side, although the knee reflex was absent. Periodic spinal drainages were done; in spite of this the pressure rose gradually. As much as 30 cc. of bloody spinal fluid was removed at one time. There was a gradual increase in the pulse and respiratory rates. Fractures of the ribs were discovered at this time, and treatment for these was given. In forty-eight hours

respiration became exceedingly rapid and labored, and the patient was put in an oxygen tent. On the third day the temperature suddenly rose to 106 F., with a respiratory rate of 40 and a pulse rate of 120. Gradually the respiratory rate rose to 50 and 60, and the patient died on June 20, 1931.

Pathologic Examination.—There was a large subarachnoid hemorrhage covering both hemispheres of the brain. Moderately severe edema was present. A frontal section of the brain showed occasional petechial hemorrhages, mainly in the white matter.

Microscopic examination revealed considerable bleeding within the subarachnoid space, with a reactive meningitis in the form of beginning proliferation of the

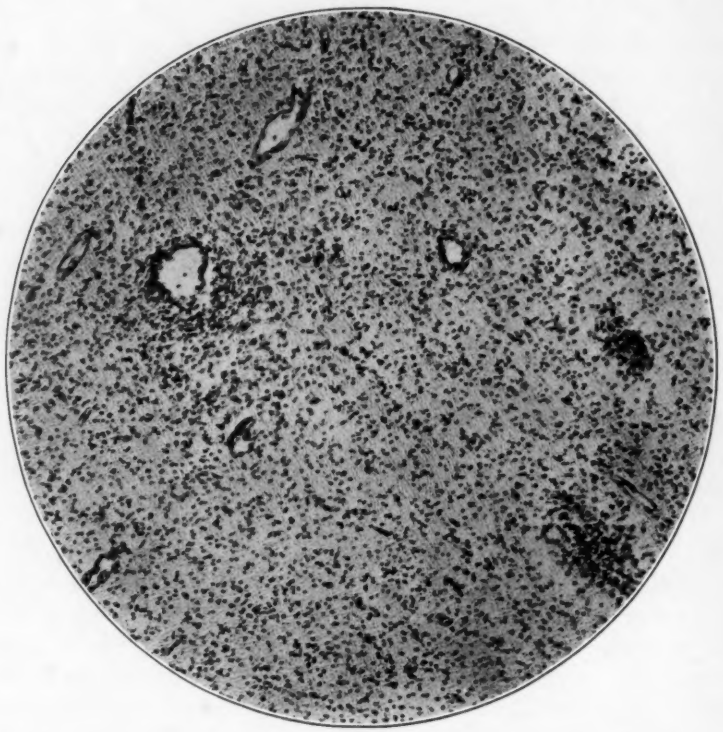

Fig. 6 (case 2).—Subcortex, showing petechiae, with general gliosis and perivascular phagocytic accumulations. Toluidine blue stain.

connective tissue elements as well as an outpouring of phagocytic cells, most of which contained blood débris. In some areas of the cortex, close to the meninges, hemorrhage and dissolution of tissue had occurred. Here the very beginnings of pial cortical adhesions could be observed. In the cortex and subcortex there were numerous petechial hemorrhages, most of which showed degeneration of the blood elements with liberation of blood pigment (figs. 5 and 6). As a consequence, there was necrosis of tissue in which the hemorrhage had occurred. Gliosis was just beginning to appear.

Much edema was present in the cortex and subcortex. This could be deduced from the presence of large spaces outside the blood vessels, in what are usually

referred to as the perivascular spaces of His. The smaller blood vessels stood out prominently as a result not only of the swelling of the lining cell but also of the intense congestion of even the smallest capillary.

One interesting feature in this case, which has been noticed particularly in other cases of traumatic hemorrhage, was the presence of blood pigment within ganglion cells (fig. 7). This was evident not only around small petechial areas, but also was seen particularly in the ganglion cells of the second and third cortical layers in areas in which the hemorrhage within the subarachnoid space was marked. There was present at the margin a great glial increase, particularly of the glia cells peculiar to this area, which are termed marginal glia.

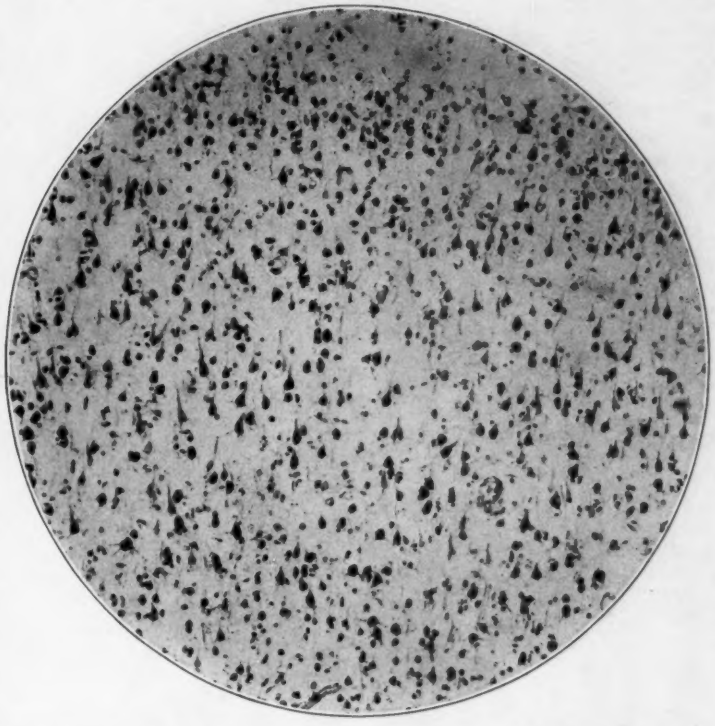

Fig. 7 (case 2).—Cortex near hemorrhage showing the presence of blood pigment in the ganglion cells. Toluidine blue stain.

Comment.—This case, in which the patient lived three days after severe head trauma, showed the clinical features of subarachnoid hemorrhage with gradually increasing edema of the brain. Pathologically, there was marked and diffuse subarachnoid hemorrhage, which had lasted long enough to produce beginning adhesions to the cortex at various points. Numerous petechial hemorrhages were present, mainly in the subcortex. The larger hemorrhages were in the cortex; some of them were in communication with the subarachnoid space. There was also blood pigment within ganglion cells.

CASE 3.—*History.*—G. W., a white man, married, aged 49, a conductor on a street car, was brought to the dispensary in an automobile in an unconscious state on Oct. 25, 1931. It was said that he had been struck by an automobile four blocks from the hospital. He had been rendered unconscious immediately and was bleeding from the left nostril and left ear. He was assigned to the neurosurgical service of Dr. Temple Fay.

Examination.—The patient was unconscious and reacted only to painful stimuli. There was no weakness in any of the limbs. There were multiple lacerations of the scalp in the frontoparietal region. There was bleeding from the nose and ear.

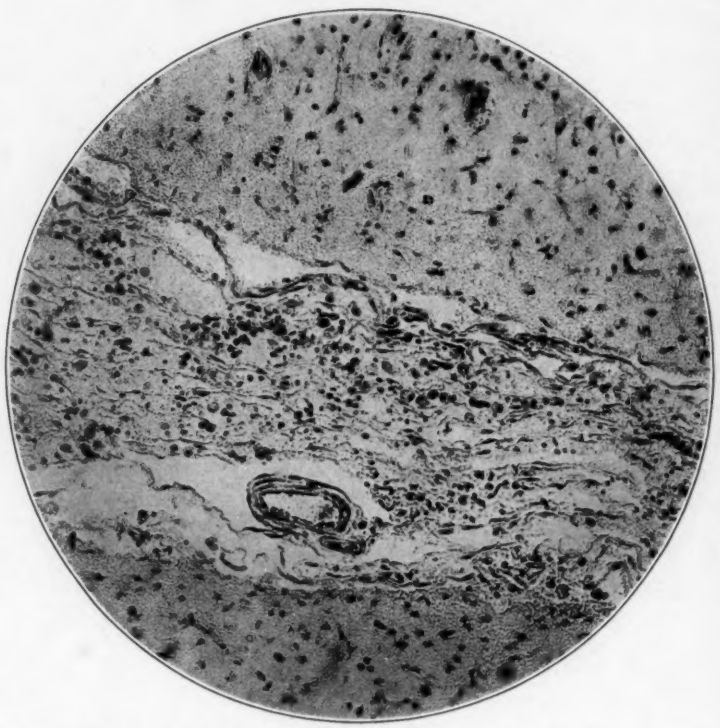

Fig. 8 (case 3).—Subarachnoid meshwork containing free blood and phagocytes. Fibroblastic proliferation is in progress. Marginal gliosis is very evident. Toluidine blue stain.

The pupils were about equal in size; the right reacted sluggishly, but the left was irregular and did not react to light. There were subconjunctival hemorrhages. The blood pressure was 110 systolic and 40 diastolic. Within a few hours after admission, the left eyelid became closed as the result of extreme ecchymosis, with weakness of the right side of the face. The pupils became very small, but were approximately equal in size and did not react to light. The reflexes throughout the body were prompt and equal on the two sides. The Babinski sign was elicited on the right side. A depression was felt in the outer table of the skull in the left frontal area. Spinal puncture yielded bloody fluid; the initial pressure was 10 mm. of mercury; 40 cc. of fluid was removed.

Course.—The patient was treated for shock; gradually the blood pressure rose until it reached 140 systolic and 76 diastolic.

On the second day, by spinal puncture 20 cc. of bloody spinal fluid was removed; it seemed to be less bloody. The patient was able to swallow on this day. On the third day in the hospital the patient's condition was not good. Râles were present in the chest, and the temperature became elevated. At 10 p. m. on the third day the patient died. Spinal fluid cell counts had shown red blood cells, originally of over a million, which decreased to 170,000 on the second day and to 24,000 on the third day.

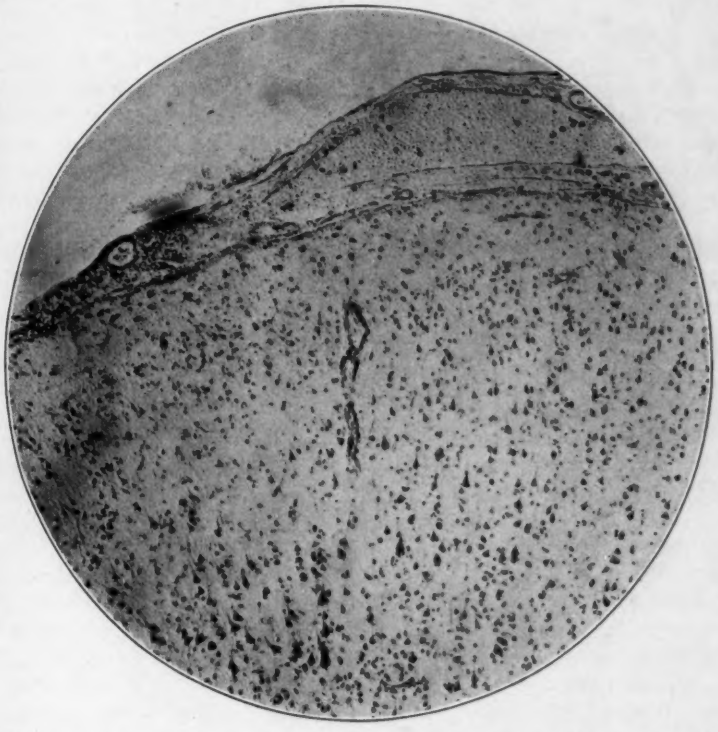

Fig. 9 (case 3).—Cortex, with acellular area (*Verödung*) near the cortical margin, with adherence of the pia to the cortex. Marginal gliosis is present. Toluidine blue stain.

Pathologic Examination.—Grossly, the brain showed definite evidences of severe trauma. Much edema was present, with thickening of the membranes over the anterior half of the brain, due mainly to the presence of free blood in the subarachnoid space. The right frontomotor area showed laceration with hemorrhage into the brain substance, some of which was being poured out into the subarachnoid space. The undersurface of the left frontal lobe also showed evidences of considerable laceration, and blood was suffused into the tissues and into the subarachnoid space. The tip of the left frontal lobe also showed laceration and bleeding, and there was evidence of a focal hemorrhage over the left lower motor area. There was no

evidence of hemorrhage into the pons. The ventricular system was not dilated Petechial hemorrhages were noted throughout the entire subcortex, as well as maceration of the brain substance in the frontal and motor areas.

On microscopic examination the subarachnoid space was found to contain free blood, much of which had already undergone disintegration. For the most part it was collected in the sulci. There was a reaction in the subarachnoid space consisting of collections of cells of phagocytic nature, most of which were collected perivascularly around many of the medium-sized blood vessels within the subarachnoid space. A beginning increase in connective tissue could also be made

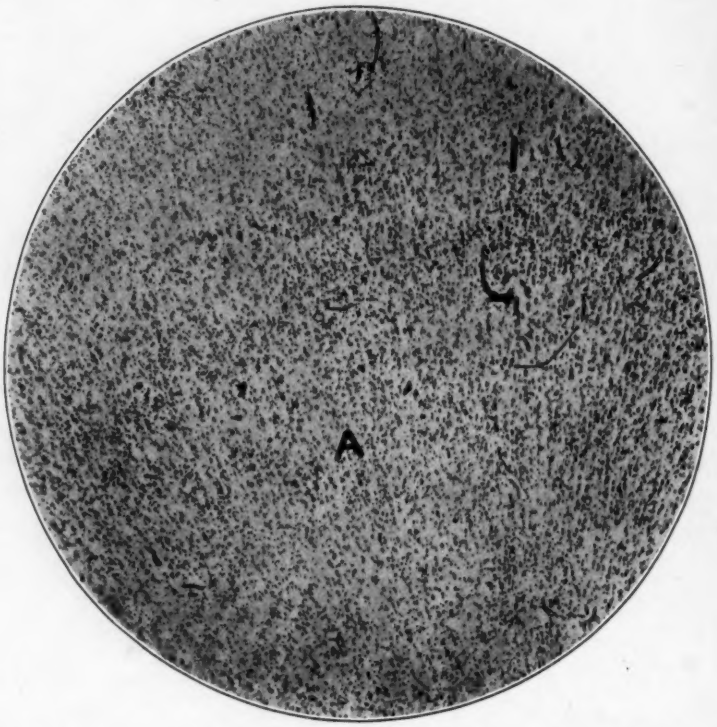

Fig. 10 (case 3).—Cortex, showing large area of *Verödung* (acellular focus [*A*]) in the third cortical layer with glial increase but without vascularization and without reaction about it. Toluidine blue stain.

out, and young fibroblastic elements were scattered diffusely throughout, with beginning adherence to the cortex in places. In one or two sulci (fig. 8) there were some free blood within the meshes of the pia-arachnoid, newly formed connective tissue elements and a great number of scattered phagocytic cells of the macrophagic type, some of which contained whole red blood cells engulfed within their bodies.

For the most part the general architecture of the cortex was not disturbed to any degree. Throughout the cortex, however, one found areas (fig. 9) in which the ganglion cells had partially or completely disappeared, and the tissue in these

areas showed evidences of a coagulation necrosis. Many were deep within the substance of the gray matter; others were superficial and in relation to the meninges. They showed usually a mild increase of glia and evidences of beginning vascularization. For the most part they were extremely small, but one or two very large areas were uncovered (fig. 10) which took in practically the entire width of the cortex. In areas of this sort, there was vascularization in and about the focus. All the ganglion cells had not completely disappeared from the area; those remaining showed evidences of severe ischemia with shrinkage and tortuosity of their processes. The glia in these larger areas showed mild increase. Universally throughout the cortex there were evidences of so-called endarteritis of the small vessels.

One characteristic feature of the cortex was a high degree of edema, which was easily recognized grossly; there was dilatation of the perivascular spaces, particularly the so-called space of His, and much rarefied tissue was visible for a considerable distance around many of the blood vessels. The ganglion cells did not show spaces around them. As the tissue was cut immediately after removal from the body and pieces were placed in alcohol, with gradual dehydration, the rarefaction around the blood vessels was taken as evidence of the intense edema. This verified the gross signs of generalized edema.

Another change, which was practically universal throughout the cortex, was the so-called marginal gliosis. Over the entire brain one noted an increase in the glia, and particularly in those areas in which blood had collected (fig. 8). Very few of the areas of the brain were exempt from this process. The entire cortex seemed to have a much greater glial content than is usually seen in normal patients of the same age. No areas were found in which actual softening of tissue had occurred, with accumulation of gitter cells. The process seemed to be entirely one of coagulation necrosis rather than a rapid vascular occlusion.

The subcortex showed in general the same type of involvement as the cortex. It should be mentioned particularly that the edema around the blood vessels seemed to be much greater than that in the gray matter, and the small vessels showed greater endothelial swelling than was present in the cortex. There had been a great increase in the astrocytes of the subcortex, as shown in Cajal preparations. Here, also, were areas in which the usual oligodendroglia picture was in the background, and rarefaction of tissue and an increase in astrocytes were to be seen.

The cerebellum showed the same general type of change that was present in the cerebrum, with congestion, edema and an occasional petechial hemorrhage, but without the acellular areas (*Verödung*) that were found scattered throughout the cortex.

Comment.—In this case, in which the patient died three days after a severe head injury, there were clinical evidences of subarachnoid bleeding with a marked increase of intracranial pressure. At autopsy, a great deal of maceration of brain tissue was found to account for the outpouring of blood into the meninges. These are not unusual findings in severe head trauma, and it is not unusual to find the maceration on the undersurface of the frontal and temporal lobes, in places where the brain does not have a thick waterbed. The explanation for the blood in the spinal fluid is not difficult, because there is a direct connection between the macerated tissue and the subarachnoid space.

Were these the only findings in the brain, the case would not have attracted much attention. The unusual feature was the occurrence throughout the cortex of areas of coagulation necrosis, similar to that in infections, toxemias and poisonings with various substances, such as carbon monoxide and lead. Areas of this sort have been interpreted by Spielmeyer [43] and his school as due to vascular spasm, even when they have been present in cases in which embolic phenomena have occurred. In our case the lesions were comparatively recent; while this patient did have arteriosclerosis, and it is possible that they could have occurred on the basis of a vascular sclerosis, still the occurrence of the great edema was a factor that must not be passed over too lightly. It is our belief that as the result of the gradually increasing edema these areas developed from gradual obstruction to the circulation.

Case 4.—*History.*—H. O., a Negress, aged 31, who was admitted to the Temple University Hospital, in the service of Dr. Temple Fay, had taken a large amount of alcohol two weeks before, ostensibly for the relief of a "grippal" infection. She became intoxicated and as a result fell down a flight of stairs. She was unconscious for just a few seconds. She remained in bed for the rest of that day, but was up and about and able to do her household duties the next morning. She began to complain of headache and was slightly nauseated, but did not vomit until several days later. For about four days she was able to do some of her household work, but was then compelled to go to bed because of a severe headache and a temperature of 101 F. The symptoms gradually increased; two days before admission she began to talk "foolishly." There had been no convulsions up to that time, but on the evening before admission she became stuporous and was convulsed.

Examination.—The patient was well developed; she was stuporous and her breathing was stertorous. The left pupil was three times the size of the right; both were stationary to light. There was a slight nuchal rigidity, with complete absence of the deep reflexes. There was neither a Hoffmann nor a Babinski sign. There was a bilateral Kernig sign. The heart sounds were of good quality, although there was a slight arrhythmia. The lungs showed definite moisture. The temperature on admission was 98 F.; the pulse rate, 76, and the respiratory rate, 26; the blood pressure was 128 systolic and 180 diastolic. A spinal puncture was done immediately. The initial pressure was 11 mm. of mercury. A faintly yellowish spinal fluid was removed. A few hours later, because the patient did not respond to ordinary treatment, another puncture was done and the pressure was found to be 6 mm. The fluid was still xanthochromic. Insulin was given because of glycosuria. The temperature, pulse rate and respiratory rate continued to increase until death, twelve hours after admission.

The patient had had numerous miscarriages and had been given a great deal of medication intravenously. The Wassermann reaction of the spinal fluid was negative. The urine contained 1.3 per cent sugar, with much albumin. The spinal fluid contained 5,080 red blood cells; the white blood count was 24,800.

Pathologic Examination.—Grossly, the brain was small but moderately edematous. There was very mild staining of the pia-arachnoid. Definite evidence of pressure was shown by the herniation of the uncus through the incisura. No definite

43. Spielmeyer, W.: Histopathologie des Nervensystems, Berlin, Julius Springer, 1922.

pressure cone was present through the foramen magnum. The basal vessels were small and bluish, and showed no evidences of atheroma. On frontal section the outstanding feature was the edema and mild congestion. The ventricles were not greatly dilated. No hemorrhages or other evidences of gross lesion were determined.

On microscopic examination the meninges showed a definite increase in the cellular elements within the subarachnoid space. These were mainly phagocytic cells, which were for the most part perivascular and contained pigment. There was a fibrosis out of proportion to the age of the patient. One occasionally found areas in which the meninges had become adherent to the cortical margin. The blood vessels within the subarachnoid spaces showed marked thickening and hyalinization of the walls.

Nowhere in the cortex was there any evidence of trauma. The changes were: (1) marginal gliosis, already described; (2) rather thick walls of the blood vessels, similar to that in the meninges, a feature not related to the trauma. The small capillaries stood out as the result of swelling of the lining cells. There was also a fairly uniform gliosis, but not to an excessive degree, and this too could be considered in proportion to the arterial changes. The large blood vessels showed practically no change. Congestion was prominent throughout the cortex but was, however, not out of proportion to what is usually seen in subarachnoid hemorrhage.

In the subcortex there was evidence that some degeneration had taken place, as shown by perivascular gliosis of pigment-containing phagocytic elements; still one did not find petechiae or areas of softening.

Comment.—This case is of interest in that the patient, as the result of acute alcoholism, fell and sustained a head trauma. There was a very short period of unconsciousness, but the patient was able to do housework by the next day and continued to do some work for about four days. As the result of an elevation of temperature and the development of headache, the patient was compelled to go to bed on the fifth day. It was not, however, until mental symptoms developed, with a convulsive attack, that the patient was rushed to the hospital and suspicion was entertained that the injury had been severe. On admission, the spinal fluid was xanthochromic. A clinical picture similar to that seen in spontaneous subarachnoid hemorrhage occurred, with symptoms suggesting localization in the hypothalamus. At autopsy, evidences of an old subarachnoid hemorrhage were found, with beginning adhesions to the cortex in occasional areas throughout the brain.

This case illustrates the effect of a comparatively mild trauma as the cause of bleeding, which may be so slow that no focal symptoms are produced. From the history one can determine easily that this patient suffered a slight meningeal hemorrhage at the time of the original trauma. Death occurred from progressive hyperthermia.

While syphilis was suspected in this case, because of the history of numerous miscarriages and intravenous medication, no evidence of this disease was found at autopsy.

CASE 5.—*History.*—E. D., a white woman, aged 75, was admitted to Temple University Hospital, in the service of Dr. Fay, in a semistuporous condition. She had fallen down a flight of stairs. She had abrasions and contusions in the right frontal region, with an ecchymosis around the right eye and laceration of the right ear. There were no palpable fractures of the skull. There were possible fractures of the radius on both sides.

Examination.—On admission, the patient was stuporous. The pupils were in middilation and equal, and reacted sluggishly to light. The upper extremities were in splints, but the lower extremities showed an exaggeration of reflexes, without a Babinski or Kernig sign. There was moderate stiffness of the neck. A spinal puncture was done shortly after admission. The pressure was 6 mm. of mercury; the fluid was slightly blood-tinged.

Course.—With repeated spinal puncture and moderate dehydration the patient reacted fairly well and was able to answer questions and obey commands. Within twenty-four hours the rigidity of the neck gradually increased. There was much clotted blood in the right nostril. There was no bleeding from the ears. Two days after admission the mental condition seemed to undergo a complete change. Roentgen examination had shown bilateral Colles fractures. Within the next five or six days the patient was irrational most of the time. On the eleventh day after admission, râles appeared at the bases of both lungs, with dulness on percussion. The patient died on the twelfth day after admission from hypostatic pneumonia.

From the laboratory standpoint the urine had been essentially normal. The blood sugar was 113.6 mg. per hundred cubic centimeters; otherwise the blood was normal. The spinal fluid contained 30,000 blood cells.

Pathologic Examination.—Grossly, the brain was of usual size, but with a great amount of edema, cortical atrophy and opacity of the pia-arachnoid over the anterior half of the brain. There was no evidence of free blood within the subarachnoid space. The basal vessels showed marked atherosclerosis.

Frontal section revealed a moderate dilatation of the ventricles, with no gross hemorrhage within the brain substance. In the brain stem, minute petechial hemorrhages were present.

Microscopically, the large blood vessels showed marked intimal thickening, with degenerative changes. The basilar vessels showed, in addition to the changes in the intima, definite fibrosis and narrowing of the media, with the presence of round cells, mainly of the lymphocytic type, in the adventitia. The smaller blood vessels, not only within the subarachnoid space but also within the brain substance, showed marked swelling of the walls with hyalinization.

Within the subarachnoid space was a small amount of free blood, most of which had already reached the stage of disintegration with the formation of a brownish pigment, much of which was already contained within phagocytic cells. The blood vessels within the subarachnoid space were all intensely congested.

There was marked congestion of all blood vessels of the cortex, with numerous perivascular hemorrhages. There was a great loss of ganglion cells, as a rule, throughout the cortex; this was not out of proportion to the sclerosis which was present. Throughout the cortex were areas in which perivascular accumulations of pigment-containing cells were found. Throughout the cortex and subcortex were comparatively recent petechiae, some of which were beginning to show regression with secondary gliosis. No great amount of edema was present, although there was definite dilatation of the perivascular spaces, particularly in the subcortex.

The ganglion cells showed nothing unusual. There had been much glial increase throughout the cortex, particularly in the marginal layer, but this again was probably not greatly out of proportion to the sclerotic changes.

Comment.—In this case, an intense arteriosclerosis was present throughout the nervous system. The cortex showed the type of change usual in arteriosclerosis; in addition, there was bloody débris within the subarachnoid space, most of which had been present long enough to be engulfed within phagocytic cells. The cortical changes in general were in harmony with the age of the patient but, in addition, there were scattered petechiae in both cortex and subcortex. The traumatic evidences in the brain were not outstanding, and one could easily have overlooked such evidences. In the medulla and pons, however, were numerous petechial hemorrhages, many of them perivascular; many were large enough to obscure the relationship to the vessels. They were old enough to bring them in relation to the time of trauma and were not agonal.

Case 6.—*History.*—J. G., a white man, aged 68, an inmate of the Masonic Home, who had been active prior to the injury so far as could be learned, had shown no symptoms of any sort prior to the accident. He had been married, but it was not known whether there were any children. He was struck by an automobile on Nov. 27, 1930, at 9 p. m. The accident occurred in front of the Temple University Hospital, into which he was taken immediately. He was treated for shock and seemed to react. There were fractures of the right arm and right leg.

Examination.—When the patient was admitted to the neurosurgical service of Dr. Fay, the pupils did not react to light; they were irregular and unequal, the left being larger than the right. The patient was sweating profusely and breathing with considerable difficulty. The temperature was 96.2 F. by axilla; the pulse rate was 112; the blood pressure was not obtainable. By spinal puncture a small amount of bloody fluid was obtained. Artificial respiration was started, but the patient died at 9: 50 p. m., exactly fifty minutes after the injury.

Pathologic Examination.—The cause of death was rupture of the liver. On gross examination, the brain, aside from mild subarachnoid bleeding, presented only very mild evidences of contusion. Microscopic examination showed mild hyperplasia of the meninges, with proliferating fibroblasts in great numbers, free blood, considerable edema, marked lymphocytic and plasma cell infiltration of the meninges and a comparatively normal cortex. The inflammatory infiltrate was concentrated mainly epicortically, as is seen in dementia paralytica, rather than spread diffusely as one finds in tuberculosis of the meninges. For the most part, the cells were of the lymphocytic type, but with some admixture of plasma cells.

While in general the large vessels showed only mild atheroma, the medium-sized vessels showed mainly hyalinization of the media, but in some the typical picture of the Heubner type of endarteritis was present.

Comment.—The patient in this case, a man, aged 68, who was considered normal for his age and was without previous symptoms, was struck by an automobile and died in less than one hour from rupture of the liver and shock. Autopsy revealed fully developed meningovascular syphilis.

The importance of this case must not be underestimated. Medicolegally, the man might have had clinical evidence of a cerebral pathologic process shortly after recovery from the acute traumatic symptoms. It is possible that, as a result of the cerebral trauma, the process within the cranial cavity might have become more active and might have produced symptoms to attract attention to the underlying condition. Since the patient died within an hour after the accident, it is evident that the trauma itself cannot be brought into relation with the cerebral pathologic process.

CASE 7.—*History.*—P. F., a white man, aged 63, was admitted to the Temple University Hospital, in the service of Dr. Fay, on Jan. 24, 1932, and died on Jan. 27, 1932. He was hit by an automobile four blocks from the hospital and was immediately rendered unconscious.

Examination.—The patient was deeply comatose, with rigidity of the neck and positive Kernig sign. His breath was alcoholic. The right pupil was larger than the left; both were irregular and fixed to light. Nystagmus to the left was present whenever attempts were made to move the eyes to the left. There was dried blood in the left naris. The ears were clear. The patient had a tendency to look to the right. The deep reflexes were absent and no pyramidal tract signs were elicited. Spinal puncture, shortly after admission, yielded bloody fluid with a pressure of 4 mm. of mercury.

There was a marked contusion of the chest; roentgen examination showed a fracture of the ribs from the fourth to the eleventh, inclusive. There was a demonstrable fracture of the skull. The right lung was collapsed as the result of puncture by a fractured rib, with resulting pneumothorax.

After the head was shaved a rather large laceration of the scalp was found. This was sutured and a drain was inserted.

Course.—Repeated spinal punctures were done during the three days while the patient was in the hospital. There was no unusual increase of pressure at any time, the highest being 8 mm. of mercury. The fluid was always bloody; many polymorphonuclear cells were present in the fluid obtained within the last forty-eight hours of the patient's life.

Laboratory examination showed comparatively normal urine. The Wassermann reactions of the blood and spinal fluid were negative. The blood contained: sugar, 156.8 mg. per hundred cubic centimeters, and urea, 20 mg. A culture of the spinal fluid yielded a greenish organism occurring mostly in short chains.

Pathologic Examination.—Grossly the brain was rather small and mildly edematous, with a great deal of subarachnoid hemorrhage, especially over the left frontal pole. The temporal poles were also covered by a considerable amount of blood. The basal vessels were whitish, but gave no evidence of atheroma.

The right cerebellar hemisphere showed marked maceration of tissue with bleeding into its substance and into the neighboring subarachnoid space.

On microscopic examination, the pia-arachnoid was found to be greatly dilated as the result of the presence of a considerable amount of free blood, as well as numerous polymorphonuclear cells with admixtures of small numbers of phagocytic elements. Most of the vessels within the pial meshes showed marked dilatation as the result of overfilling with blood. It was over the vertex, particularly, that the phagocytic elements were most numerous. There was a tendency, as is usual, for the macrophages to be collected at the margins of the subarachnoid space rather than in the center.

The cortex showed the features usual in both subarachnoid hemorrhage and acute meningitis. There was definite pallor of practically all the ganglion cells within the brain. This corresponds with the so-called cloudy swelling rather than with a more serious degenerative process. In the marginal layer there was an even more marked gliosis, such as is found in most irritative conditions within the subarachnoid space. Small blood vessels within the cortex were a little more prominent than usual as the result of unusual congestion as well as swelling of the lining cells. A moderate amount of perivascular edema was present, but this was by no means uniform throughout the cortical tissue. Only in an occasional area could one see an effort at penetration of the meningeal exudate into the cerebral substance adjacent to the meninges. At no point was there a very marked penetration into the cortical tissue.

Within the subcortex were found two types of lesions: 1. The usual petechial hemorrhage that occurs in so many different conditions. These were comparatively fresh, with only beginning degeneration of the blood elements, and many were perivascular. 2. Small areas of necrosis without the presence of blood elements. In these areas an effort had been made to repair the damage by means of proliferation of glia, mostly of the astrocytic type, while within the areas were occasional phagocytic elements containing remains of débris. In the immediate neighborhood of these areas there were usually blood vessels perivascularly infiltrated with phagocytic cells which had lost their pigment. The entire subcortex presented a lacelike structure as the result of edema. There had been a great increase of the astrocytic cells at the expense of the oligodendroglia. Perivascular edema was evident.

Comment.—Historically, this case presents an unusual sequence of events. The patient, who was rendered unconscious as the result of trauma, had, as the acute manifestations of the trauma, marked subarachnoid bleeding as well as numerous petechial hemorrhages and areas of necrosis within the subcortex. As the result of an infection of the subarachnoid space, evidenced by the finding of organisms within the spinal fluid during life, an acute meningeal infection was added to the subarachnoid bleeding.

The question of the entrance of the organisms into the subarachnoid space might be of considerable interest and importance. While it is possible that organisms can be, and have been, introduced with a spinal puncture needle, especially in cases in which devitalization of the tissue elements has occurred, there are still two other possibilities: (1) the patient had a fracture of the bones of the skull with laceration of the scalp, and (2) infection was present somewhere in the body, with localization at a point of lessened resistance.

COMMENT

It has long been recognized that so often are there residual findings following severe head trauma, even when litigation is not in question, that some organic change must be present to account for the clinical pictures. It is important to distinguish between cases in which the head injury was severe and those in which mild trauma was only a minor

incident in the picture. Just as Mott distinguished between shell shock which was the result of actual concussion due to an explosion and that which was the result of psychic trauma, so one must endeavor to distinguish between real and feigned injury to the cranial contents. In the distinction between mild and severe cranial lesions the duration of unconsciousness is of importance, especially in the absence of complications. Symonds [22] and Russell,[27] following Trotter,[17] have taken the duration of loss of full consciousness at the time of the accident as an indication of the degree of cerebral injury. In general, it can be stated that the greater the damage to the brain the longer the unconsciousness.

That severe damage to the brain does occur is amply substantiated by the reports of many cases in the literature in which the changes were so marked following severe head trauma that actual psychoses were produced. Adolf Meyer,[35] Rosenhagen,[44] Wohlwill [37] and Jakob [38] reported histologic evidence that such psychoses were accompanied by actual changes in the brain.

When, of course, the clinical picture is marked and the histologic change evident, the inescapable conclusion is that, as the result of trauma, a pathologic change has been produced which is directly responsible for the clinical symptoms.

When, however, the mental picture is less marked and the corresponding changes in the brain are not so evident, the histologic changes can be brought out only with careful studies. It is on this type of case that our work has centered; in our cases, although severe enough to produce death in days or weeks, we have sought not so much for gross lesions of the brain, which are sufficiently well known, but rather for minute changes which might be held responsible for the type of clinical picture that is usually designated as traumatic neurosis.

Our gross findings were not unusual. The most common condition was subarachnoid bleeding. Frequently this was mild, and we were able to see at death only the terminal result of the bleeding. In the early stage of the bleeding one finds well preserved red blood cells free within the meshes of the dilated pia. Shortly after this there is a reaction in the pia, which can be recognized by disintegration of the blood elements, beginning proliferation of the fibroblasts and an outpouring of phagocytic elements. In places one finds an associated reaction in the adjacent cortical tissue, with secondary gliosis in the immediate area involved. In this way pial-cortical adhesions are formed (Bagley [45]). In many

44. Rosenhagen, H.: Ueber postkommotionelle Veränderungen im Gehirn, Deutsche Ztschr. f. Nervenh. **114**:29, 1930.

45. Bagley, C.: Blood in the Cerebrospinal Fluid: Resultant Functional and Organic Alterations in the Central Nervous System, Arch. Surg. **17**:18 (July) 1928.

cases in which these adhesive lesions have been found, convulsive seizures have been absent (Foerster and Penfield [46]).

We have intentionally omitted cases with epidural, subdural and late hemorrhages. They have already been well covered in the literature (Goroncy,[11] Eck [13] and Smidt [12]).

As regards fractures of the skull, we shall mention only what is well known to neurologists, that the absence of fractures of the cranial vault or base gives no indication of the severity of damage to the underlying brain tissue.

As for the brain itself, gross hemorrhages with maceration of tissue are comparatively common in rapidly fatal cases. Minute hemorrhages are apt to occur in the less severely injured. Edema has not been a universal finding, and the view of Apfelbach [15] that edema is not present within the first several hours, or after the third day, is probably correct. When edema does occur there is interference with the blood supply to the brain (Hoff,[47] Gildea and Cobb [48]). We have found ischemic changes in the ganglion cells and areas of focal necroses similar in every way to the areas resulting from obstruction to the minute blood vessels from any cause.[49] These lesions may be important in explaining the symptoms and signs occurring months or years after a trauma. That gross changes can occur in the brain and remain unsuspected has recently been brought to light in routine encephalographic studies in cases of head trauma. Wartenberg [2] was one of the first to stress the occurrence of unilateral ventricular dilatation as an after-result of head trauma when a neurosis was suspected clinically. We have seen meningeal adhesions to the cortex, with areas of *Verödung* in the brain in sufficient numbers to produce a ventricular change as noted by Wartenberg and others (Penfield,[42] del Rio Hortega [50] and Winkelman [51]).

As a result of the trauma itself, as well as the general ischemia which the edema produces, one would not be surprised to find a generalized gliosis similar in all respects to that due to any other cause of a

46. Foerster, O., and Penfield, W.: Traumatic Epilepsy: Structural Basis of Traumatic Epilepsy and Results of Radical Operation, Brain **53**:99, 1930.

47. Hoff, H.: Experimentelle Studien zur Frage des postkommotionellen Hirnödems, Ztschr. f. d. ges. Neurol. u. Psychiat. **129**:583, 1930.

48. Gildea, E. F., and Cobb, S.: The Effects of Anemia on the Cerebral Cortex of the Cat, Arch. Neurol. & Psychiat. **23**:876 (May) 1930.

49. Winkelman, N. W., and Eckel, J. L.: Endarteritis of the Small Cortical Vessels in Severe Infections and Toxemias, Arch. Neurol. & Psychiat. **21**:863 (April) 1929.

50. Penfield, W., and del Rio Hortega, P.: Cerebral Cicatrix: The Reaction of Neuroglia and Microglia to Brain Wounds, Bull. Johns Hopkins Hosp. **41**:278 (Nov.) 1927.

51. Winkelman, N. W.: Mechanism of Brain Injuries from a Pathological Standpoint, Ohio State M. J. **28**:429, 1932.

similar nature. This will tend to disappear gradually, just as it does during the remission stages of dementia paralytica or even in acute inflammation of the brain, as in acute meningitis. The changes in the glia in trauma have been well covered by Rand and Courville,[52] Roussy, Lhermitte and Oberling [53] and Penfield [42] and experimentally by Stevenson,[54] so that it need not be discussed here (Tschistowitsch,[55] Knauer and Enderlen,[56] Wilson,[57] Penfield and Buckley [58] and Wortis [59]).

The changes in the small vessels are of interest and probably important in the production of secondary changes. We frequently saw a prominence of the small capillaries out of keeping with the age of the patient. This is probably the result of general anoxemia of the brain from the resultant edema. Acellular foci may result from this condition alone.

The ganglion cells show only the effects of the general ischemia. In one of our cases, in which hemorrhage had taken place, the blood pigment was present within the cytoplasm of the ganglion cells (Spielmeyer [43] and Jakob [38]).

In the subcortex, petechiae are fairly common. With these one finds small necrotic areas even in the absence of free blood, similar in every way to those seen in purpuric brains from a multitude of causes. The cerebellum in one of our cases was of interest in view of the frequency of posttraumatic dizziness and sometimes even disturbances of gait. In this case the softening in the cerebellum differed in no way from the softening seen from the usual vascular occlusions. In one of our cases petechiae occurred in the pons and medulla, a not unusual finding in severe traumatic cases. The basal ganglia have presented nothing unusual.

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52. Rand, C. W., and Courville, C. B.: Histologic Changes in the Brain in Case of Fatal Injury to the Head: III. Reaction of Microglia and Oligodendroglia, Arch. Neurol. & Psychiat. **27**:605 (March) 1932; IV. Reaction of the Classic Glia, ibid. **27**:1342 (June) 1932.

53. Roussy, G.; Lhermitte, J., and Oberling, C.: La névroglie et ses réactions pathologiques, Rev. neurol. **1**:878 (June) 1930.

54. Stevenson, L. D.: Head Injuries: Effects and Their Appraisal; II. The Rôle of the Microglia, Arch. Neurol. & Psychiat. **27**:784 (April) 1932.

55. Tschistowitsch, T.: Ueber die Heilung aseptischer traumatischer Gehirn-Verletzungen, Beitr. z. path. Anat. u. z. allg. Path. **23**:321, 1898.

56. Knauer and Enderlen: Die pathologische Physiologie der Hirnerschütterung nebst Bemerkungen ueber verwandte Zustände, J. f. Psychol. u. Neurol. **29**:1, 1922.

57. Wilson, R. B.: Brain Repair, Arch. Neurol. & Psychiat. **15**:75 (Jan.) 1926.

58. Penfield, W., and Buckley, R. C.: Puncture of the Brain: The Factors Concerned in Gliosis and in Cicatrical Contractions, Arch. Neurol. & Psychiat. **20**:1 (July) 1928.

59. Wortis, S. B.: Head Injuries: Effects and Appraisal; I. Experimental Studies of Induced Convulsions and Ventricular Distortion in the Cat, Arch. Neurol. & Psychiat. **27**:776 (April) 1932.

We have described a case in which death occurred as the result of acute meningitis, probably from contamination at the time of the spinal puncture. In cases in which direct opening into the meninges occurs, one can easily understand the mechanism of the meningeal inflammation. In our case, in which no such communication existed, and in which the meningitis began immediately after the spinal puncture, the relationship seems fairly clear.

One need, therefore, not be surprised, in view of the histopathologic changes in the brains in most of the cases of severe head trauma, to find clinical symptoms in direct relation to the degree of damage. While psychoneurotic manifestations occur and cannot be denied, still, in cases in which unconsciousness occurs at the time of the injury and lasts over a period of time there is sufficient damage to the brain in the form of gross and minute lesions to account for symptoms that may be permanent. On the other hand, with mild trauma and without unconsciousness, the symptoms cannot be explained on the basis of permanent histologic changes in the brain.

SUMMARY AND CONCLUSIONS

1. A histologic study of a selected group of cases of brain trauma has been undertaken.

2. Occasionally, a definite pathologic process is going on at the time of death which is not the result of trauma but which might have become manifest or more active following trauma. As an example, we have described a case of chronic syphilitic meningitis in a patient in whom death occurred less than one hour after injury.

3. Subarachnoid hemorrhage is the most common gross lesion. Large and small hemorrhages within the brain substance are comparatively common.

4. The onset of edema shortly after an injury increases the damage to the brain. As an illustration of this we have described the case of a patient who lived for three days after the accident with multiple areas of softening throughout the cerebral cortex, differing in no way from those seen in arteriosclerosis, severe infections and intoxications.

5. The development of pial-cortical adhesions could be traced from the beginning as the result of subarachnoid hemorrhage and secondary reaction.

6. Sufficient evidence has been found in the brains of patients with severe head trauma, especially those in whom unconsciousness had been prolonged, to convince us that most of these persons have organic changes in the brain, the result of the trauma. These changes explain the posttraumatic symptom complex usually characterized as "traumatic neuroses."

DISCUSSION ON PAPERS BY DRS. STRAUSS AND SAVITSKY AND DRS. WINKELMAN AND ECKEL

DR. WILLIAM G. SPILLER, Philadelphia: The importance of thorough study of the clinical aspects of head trauma has been ably presented by Drs. Strauss and Savitsky, and the histopathologic aspects have been presented by Drs. Winkelman and Eckel. The latter authors have preferred to devote their attention largely to the minute changes in the brain—those that may readily escape detection and yet are of great importance. Lesions such as fracture of the skull and epidural, subdural, subarachnoid and intracerebral hemorrhage are familiar to all physicians, but the following conditions are less so: mild congestion; mild degrees of edema; multiple petechial hemorrhages; diffuse or limited gliosis; new blood vessel proliferation; minute areas of rarefaction; phagocytosis of blood pigment by ganglion cells; ischemic changes in the ganglion cells; disseminated minute areas of softening in the cerebellum with gitter cells; disappearance of the Purkinje cells; cellular loss in the dentate nucleus; alterations in the subarachnoid space such as small collections of blood corpuscles, especially in the sulci; infiltration of phagocytic cells in the pia-arachnoid; beginning proliferation of connective tissue with young fibroblastic elements, and adhesions of the pia to the cortex.

These authors attribute the headache following brain trauma, at least in part, to the pial adhesions, and they think that it may be relieved through rupture of the adhesions by injected air. Recent studies by Hassin, Penfield and others on the presence of nerve fibers following the blood vessels of the pia and even extending into the cortex may explain the headache on the basis of irritation of these fibers in the adhesions.

It does not seem difficult to accept the statement that lesions such as these may be fruitful in the production of psychoses or of any of the symptomatic disturbances of the brain that follow head trauma, and yet one dare not conclude that every disturbance of nerve cell function is registered by detectable histologic changes.

DR. LOUIS CASAMAJOR, New York: Dr. Savitsky's presentation has raised one question: What is a normal encephalogram? One sees so many encephalograms in which this or that appearance is pointed out as abnormal that one wonders how these anomalies are recognized. Up to the present time no norms have been established, as too few controls have been studied. I am not convinced that the findings shown in this presentation are really due to trauma. I wonder what these patients would have shown had the encephalograms been made a week before the injury rather than immediately afterward. I do not think that enough is known at present to enable one to conclude from a single finding in the encephalogram that a condition is due to any particular cause. I have recently seen a case of tumor of the brain, proved at operation, in which the encephalogram was practically normal according to the interpretation of physicians who are used to reading these films. I am wondering what a normal encephalogram is.

DR. S. E. JELLIFFE, New York: There is little question but that everybody here is interested in the problem which has been presented so fully and so lucidly. The majority of the members of this association are called on to exercise a special function in society in endeavoring to evaluate different factors following such injuries. For a number of years it has been a special interest of my own to try to find out how much the unconscious knows about injuries inside of the body. One must not for a moment assume that in such a study any depreciation of the usual methods of getting at the facts is conceived of. All due valuation and

recognition are given to micro-anatomic dissection and to microneurologic dissection; but with the newer methods of micropsychic dissection is one going to get any information that may be of service in determining the relative not "either or" but "and or" of the situations involved?

It would be presumptuous to offer any hard and fast formulas, but certain tentative formulations concerning what the unconscious knows about these things and how to get at them have been growing up in my mind. I should state my attitude all too dogmatically or all too statically by saying that in the study of the unconscious processes—chiefly, of course, by means of the dream process—to the degree to which the person projects his history of injury into the dream, I minimize the significance of actual organic changes, and as he pays less and less attention in the unconscious to the actual traumatic event and more to symbolic representations of revenge and retribution, I attribute more and more significance to the actual organic injury. Whether or not such a formulation will hold after more extensive study or not, I am not prepared to say.

Dr. Bernard Sachs, New York: I should like to say in connection with the whole subject, so as to avoid one danger that neurologists seem to be steering for, that the cases that have been presented are instances of rather serious head injury and I do not think there has been any doubt that organic lesions have followed such injuries.

However, before one absolutely dismisses the idea of anything like the picture of traumatic neurosis, an important question and one that has great practical value, particularly in medicolegal experience, is what happens in the far greater number of cases of rather slight injuries to the head and to the spine, in which there is a remarkable similarity in the claims made and in the symptoms that are supposed to follow. Whether or not there is distinct organic change in those cases is still open to discussion. At all events, I think it should be allowed that there may be something in the form of a general neurosis or of neurotic symptoms not necessarily based on actual organic changes which frequently follows mild injury to the brain or to the spinal cord. The mistake that should be avoided is the insistence that in all cases of head injury there are the changes that have been shown here. It has been valuable to see the evidence of the changes that occur, particularly in such cases as Dr. Winkelman presented, but one should not jump at the illogical conclusion that there are organic changes in all cases of skull trauma. I am inclined to feel that evidence is lacking that there are organic changes following even the milder forms of skull trauma.

Dr. Peter Bassoe, Chicago: Dr. Winkelman spoke of phagocytosis of blood pigment by ganglion cells. Does he mean that the ganglion cells themselves are phagocytes or that other, humbler cells have carried the blood pigment into the ganglion cells and then perhaps perished, leaving the pigment there?

Dr. Israel Strauss, New York: If any one today reads Oppenheim's monograph on traumatic neurosis, he will find that many of the cases cited by Oppenheim were organic. Oppenheim did not have at his command many of the signs and symptoms of organicity which physicians now have at their disposal. Consequently many of his cases would today be regarded by any neurologist as organic and not functional.

Unfortunately, one has no knowledge of the pathologic changes many years after head trauma.

We chose for presentation here encephalograms that we know, from our experience and also from what has been written in the literature, are abnormal. We have not taken as a normal encephalogram the criterion which Foerster and his

school have adopted, for the reason that what they called normal is a little too limited in scope. We think that there are more variants than they admit. But even from our liberal point of view, we consider that the encephalograms which we have shown designate abnormality.

I must take exception to Dr. Winkelman's statement that head trauma must be severe to cause organic changes. This paper is based on a study of almost seventy-five cases. Many of the patients came to me from the Industrial Commission of the State of New York, some of them with symptoms of from two to six years' duration, all of them complaining constantly and all of them considered by the insurance company as exaggerators of symptoms or, occasionally, as malingerers. Of all the patients that I have seen, only two could be proved to be malingerers, and neither of these was suffering from head trauma.

DR. N. W. WINKELMAN, Philadelphia: Dr. Sachs' warning is in order because it cannot be denied that there are in general two main groups of patients: those with definite and severe head trauma and those in whom brain injury either has not occurred or has been minimal. Our presentation today concerns the first group only, and in them we have found sufficient to account for the posttraumatic symptoms and for the encephalographic findings shown today and at former meetings.

In reply to Dr. Bassoe's question about the so-called phagocytosis of the blood pigment by ganglion cells, we found in two or three of our cases that the ganglion cells in the neighborhood of old hemorrhages contained blood pigment. This is not an artefact, as has been thought by some. The mechanism of this process is unknown because ganglion cells are not motile and usually not phagocytic. We mentioned this only because it may be evidence of a functional derangement of the ganglionic elements.

BULBOCAPNINE

EFFECT ON ANIMALS WITH LESIONS OF THE CENTRAL NERVOUS SYSTEM

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AND
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The typical picture of catalepsy as produced by moderate doses of bulbocapnine is now widely known; for the present it is sufficient to recall that it is chiefly characterized by immobility, loss of motor initiative, resistance to passive movement, maintenance of the body in attitudes which would ordinarily be obnoxious or inconvenient and submission to the assumption of passively induced abnormal positions of the limbs. Larger doses, on the other hand, cause hyperkinesia and even epileptiform seizures.

We have been impressed by the similarity between the symptoms reported to be due to bulbocapnine and certain cataleptic phenomena observed in this laboratory as the result of the production of lesions in the upper part of the midbrain in cats (Ranson and Ingram [1]). This catalepsy, which was associated with somnolence, was characterized chiefly by a markedly plastic type of muscle tonus. It was possible to mold these cats in odd postures which they would retain for considerable periods of time. The plasticity was so marked that the limbs would retain passively induced positions involving varying degrees of extension, even when the animal was supine, with the feet free from any contact. There was marked resistance to passive movement, especially to flexion. The animals were able to right themselves, stand and walk, but were unwilling to do so under ordinary circumstances. If they were suddenly thrown off balance they could shake off their lethargy long enough to right themselves properly. When they were in the most profound stages of this catalepsy, situations such as the proximity of a live mouse, which would ordinarily arouse a normal cat to sudden activity, would elicit no interest. In less profound stages the animal might become aroused, seize the mouse and then lapse into its lethargic state with the mouse dangling from its jaws. The duration of this condition varied; in some cats it disappeared after a few days, while in others it was found to persist for weeks or even months. The lesions responsible

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1. Ranson, S. W., and Ingram, W. R.: Catalepsy Caused by Lesions Between the Mammillary Bodies and Third Nerve in the Cat, Am. J. Physiol. **101**:690, 1932.

for the development of these phenomena were found to involve the area between the third nerve and the supramamillary region bilaterally, and were chiefly centered about the point where the habenulopeduncular tracts approach the ventral surface of the brain. There was often some encroachment on the gray matter of the floor of the caudal part of the third ventricle; the tegmental fields of Forel were seriously affected, and the lesions had a cross-sectional extent sufficient to involve many of the little known but important pathways descending from the striate bodies and the hypothalamus. The great ascending pathways, especially those conveying proprioceptive impulses, were usually but little affected. The pyramidal path was never involved.

The general similarity between the catalepsy described and that reported to be due to bulbocapnine intoxication led us to believe that interesting results might be obtained from the administration of this drug to cats in which catalepsy-producing lesions had previously been made. Therefore a series of experiments was instituted in which the effects of varying doses of bulbocapnine on cats which were already cataleptic or which had partially recovered from the catalepsy induced by lesions in the midbrain were studied and compared with the results of similar procedures carried out on animals which had been subjected to other types of operations.

MATERIAL AND METHODS

In the experiments reported here bulbocapnine phosphoricum was used. This may be obtained either in solution in sterile ampules or as tablets. Both forms were utilized; the tablets were dissolved in suitable amounts of sterile water. Doses varying from 5 to 50 mg. per kilogram of body weight were administered to a series of normal cats and to a group of cats on which various operations had been performed. Altogether, twenty-three normal animals were used.

Among the cats on which the operations were performed were two in which the lumbar sympathetic chain had been removed unilaterally, two in which the left frontal lobe of the cerebrum had been excised, two in which bilateral labyrinthectomy had been performed, six in which both red nuclei had been partially or completely destroyed and eight in which catalepsy had been induced by lesions in the upper part of the brain stem.

The lesions in the brain stem were produced with the aid of the Horsley-Clark stereotaxic instrument, the use of which has been described in previous papers (Ingram and Ranson and their collaborators [1a, b]). Bipolar needle electrodes were introduced into the desired region of the brain stem, and small lesions were produced by the passage of direct current of from 1 to 3 milliamperes for from twenty to sixty seconds. It has been found possible to destroy a red nucleus with little damage to the surrounding structures by placing three such lesions in a row

1. (*a*) Ingram, W. R.; Ranson, S. W.; Hannett, F. I.; Zeiss, F. R., and Terwilliger, E. H.: Results of Stimulation of the Tegmentum with the Horsley-Clarke Stereotaxic Apparatus, Arch. Neurol. & Psychiat. **28**:513, 1932; (*b*) Ingram, W. R., and Ranson, S. W.: The Effects of Lesions in the Red Nuclei in Cats, ibid., p. 483.

rostrocaudally and so close together that they become confluent and form a single elongated lesion. The lesions causing catalepsy were produced in a similar manner but were located in the area immediately rostral to the red nuclei, often involving the frontal poles of the latter.

These animals were utilized only in the chronic stages, after all the wounds had healed completely and a state of good general health had been attained. One cat of the cataleptic group still showed noticeable signs of catalepsy when the first injection was made; the other seven were in various stages of recovery. They were no longer somnolent and were capable of spontaneous activity. All had previously displayed profound catalepsy and hypersomnia.

OBSERVATIONS

Our experiments revealed a remarkable similarity between the catalepsy produced by bulbocapnine in normal animals and the catalepsy caused by retromamillary lesions. When suitable doses of the drug were administered to cats which had previously been operated on for the production of catalepsy or for destruction of the red nuclei, the general character of the effect was not greatly altered. However, it was found that susceptibility to bulbocapnine was noticeably increased following bilateral destruction of the red nuclei and was markedly increased after the production of retromamillary lesions. In the latter instances catalepsy of great depth could be induced by comparatively minute doses.

NORMAL ANIMALS

In one normal cat 50 mg. of bulbocapnine for each kilogram of body weight was injected subcutaneously; one cat received 40 mg. per kilogram; two received 35 mg.; one, 30 mg.; two, 25 mg.; six, 20 mg.; five, 15 mg.; seven, 10 mg., and three, 5 mg.

According to Schaltenbrand,[2] small doses of from 5 to 10 mg. per kilogram cause a tendency to somnolence and occasional sympathetic effects. As the amounts administered are increased, the animals become more and more immobile and assume attitudes of flexion. The terms "flexion tendency," "flexion position" or "flexion attitude," as used in the subsequent account, may be interpreted as referring to the disposition of an animal to assume a crouched position, with the limbs flexed beneath it and the back somewhat arched. The cataleptic state is supposed to become evident with doses of from 20 to 30 mg. per kilogram, but when these are increased to 40 or 50 mg. there seems to be a period of less severe involvement which marks the beginning of the hyperkinetic stage. With progressively increasing doses this finally attains its supreme expression in epileptiform convulsions. It is emphasized by various authorities, however, that there are considerable racial and individual variations in response to bulbocapnine; this we can confirm. The two cats in our series which received 40 and 50 mg. of the drug per kilogram of body weight displayed no true hyperkinetic phenomena. Within fifteen minutes they assumed a flexion position, crouched on the floor and remained immobile. It was shortly possible to induce passively various abnormal positions of the limbs,

2. Schaltenbrand, G.: Die Beziehungen der extrapyramidalen Symptomenkomplex zu den Lage- und Bewegungsreaktionen, zum motorischen Haushalt und zu den Stammganglien, Deutsche Ztschr. f. Nervenh. **108**:209, 1929.

extreme abduction, for instance, which were retained for long periods. The animals would not assume postures involving the maintenance of extension of the limbs, however. They were capable of supporting their own weight when suspended by the forelimbs, clinging to a support for some time, or when placed like a bridge on two supports with the forelimbs drawn forward and the hind limbs drawn backward. This is the stage called "active" catalepsy by de Jong, and when these phenomena are referred to in the following account they will be designated, for brevity's sake, as "hanging" and "bridging." When the animals were placed in a supine position, resting in a trough, the head and pelvis were raised, and the spinal column was flexed, but no further attempts at righting the body were observed. When the head was pushed back and then released, it gradually returned part way to its initial position. The cat which had received 40 mg. per kilogram showed no evident change in muscle tonus, but the one to which 50 mg. per kilogram had been administered showed slight but obvious increases in muscle tonus, with resistance to passive movement of the limbs and also to stimulation of the *Stütz* reflex by pressure against the pads of the feet. There were traces of plasticity in the forelimbs. The effects of the large doses persisted in some form for several hours, but after an hour and a half one of these animals was able to release itself from a hanging position and to spit and strike when another cat was brought in immediate proximity. Neither of these cats showed any noticeable sympathetic effects, such as vomiting or loss of sphincter control, although these phenomena were often seen in cats which received much smaller doses. Considerable drowsiness was manifest at certain times; at other times there seemed to be a fair degree of awareness.

With 35 mg. per kilogram, a similar picture was obtained; these cats, however, showed distress by frequent crying. There was no excessive salivation, vomiting or loss of control of the sphincters. Immobility, flexion attitude and submission to unusual, passively induced positions of the limbs were again evident, as was the ability to suppor' the weight of the body in the odd postures mentioned. The tendency to flexion of the spine persisted when the animal was supine. There was no evidence of increased tonus of the limbs. Similar results were obtained with 30 mg. per kilogram. Twenty-five milligrams per kilogram administered to two cats produced effects which were at considerable variance. One of these animals failed to lose its motor initiative, and, although drowsy and stupid, was able to rise and move about when disturbed. The other showed more evident catalepsy, with the usual symptoms. There was no increase in the muscle tonus of the limbs, and the only evidence of excessive salivation was frequent licking of the lips. A certain amount of awareness was apparent in this animal throughout the experiment.

With doses of 20 mg. per kilogram there was also considerable variation in the behavior of the animals. Two cats failed to show complete immobility but were able to move about when disturbed. There was a tendency to drowsiness and flexion attitudes, but passively induced postures were not maintained, nor was it possible to place the animals in the supine position. Two other cats showed a fair degree of catalepsy, but this was not so marked as that produced by larger doses. There was considerable crying, but no vomiting, and only slightly excessive salivation. Evacuation of the bladder occurred in one cat. The usual symptoms were displayed, especially in regard to the crouching, flexed postures spontaneously assumed, the loss of volitional motility and the maintenance of induced

poses, including hanging in one instance. When the animals were placed in the supine position there was initial refractoriness, but later there was temporary acceptance of the attitude, and in one cat there was slightly increased muscle tonus. Two other animals showed rather good catalepsy when this dose was given.

Three of four cats which were given injections of 15 mg. of bulbocapnine per kilogram became somewhat stuporous but never displayed true catalepsy. They refused to pose in passively induced positions and were unwilling to remain supine. One of these animals salivated rather freely and vomited; the others gave no indication of disorders of this sort. One animal showed mild catalepsy, and a fifth cat treated with this dose showed unmistakable cataleptic tendencies. This animal and one other which seemed to be especially susceptible to the drug will be accorded special mention later.

Of six cats which were given 10 mg. of bulbocapnine per kilogram, five showed some distress and inertia but no real catalepsy. Two vomited; all had somewhat excessive salivation, and their discomfort was expressed by frequent cries. When at rest the usual flexion position was assumed, but the capacity for voluntary and reflex movement was retained, and there was some difficulty in holding them on their backs. These animals refused to pose, and, aside from slight drowsiness, timidity and ease of handling they could hardly be distinguished from normal animals. The sixth cat of this group reacted more strongly to the drug; some passively induced poses were retained for short periods, and it rested quietly when supine. However, through most of the period of observation it showed a fair degree of alertness and awareness, giving slight starts at sudden noises. No apparent vegetative symptoms were displayed by this animal.

With 5 mg. per kilogram, which was the smallest dose given in our series, the behavior of the animals was similar to that observed when 10 mg. was injected, except that the inertia did not have as great a depth or duration. There was the usual tendency to quietude, but there was no disposition to retain abnormal positions.

Besides these there were two cats which seemed to possess a special susceptibility to the effects of bulbocapnine. One was given 15, 10 and 5 mg. per kilogram at intervals of from ten to twenty days. With the largest dose unmistakable evidences of catalepsy were obtained, with ready submission to the various procedures involved in determining the degree of intoxication. With 10 mg. per kilogram the catalepsy was less marked but was still more pronounced than that caused by such a dose in the average cat; the same statement may be made as regards the 5 mg. dose. There was profuse salivation in each of these instances. The other cat showed profound intoxication following a dose of 10 mg. per kilogram, adopted poses readily and showed some plasticity of the limbs but no other changes in tonus. Two weeks later this cat was again given injections of 10 mg. per kilogram, and again it showed a greater reaction than did the average cat. These two animals are mentioned to illustrate the variation in susceptibility which may be found in a large group. In general, however, in our experience the average normal cat gives the best cataleptic reactions to doses of bulbocapnine ranging from 20 to 40 mg. per kilogram, with such variations as have been mentioned in the preceding paragraphs. Doses of 15 mg. per kilogram or less are not ordinarily sufficient to induce satisfactory catalepsy, the chief symptoms induced being inertia and drowsiness. That certain animals may evince affinity for the drug is shown by two cases just described.

SYMPATHECTOMIZED CATS

Two cats in which the lumbar sympathetic chains had been removed unilaterally were given 20 mg. of bulbocapnine per kilogram of body weight. Both animals proved to be somewhat refractory to this dose, and, although they manifested considerable stupor and inertia, they refused to accede to most passively induced positions. There were no changes in tonus or loss of reflexes. Both showed some excess salivation, and one vomited.

PARTIALLY DECORTICATED CATS

Two cats in which the entire left frontal lobe had been removed were given injections of 20 mg. of bulbocapnine per kilogram. Prior to the injection both of these animals showed a tendency to turn to the left in walking, but good control of the limbs had been established. When the animals were supine the right limbs tended to extension and showed somewhat greater rigidity than did the left. Shortly after the injection there was a reversal of the tendency to turn to the left; in each case the head was now turned to the right, and in walking the cat circled to the right. The right limbs seemed to become more rigid and less well controlled. Both animals were somewhat recalcitrant when efforts were made to pose the limbs, the only evidence of posing being given by the right forelimbs. Profound drowsiness was the sequel of the injection, but complete immobility was not obtained. When the animals were supine the tonus of the extensors of the right limbs seemed to be increased, and only these limbs gave any signs of plasticity. The body-righting reflexes were intact. One cat showed profuse salivation and vomiting; the other showed some loss of control of the bladder. These findings are essentially similar to those of Ferraro and Barrera in a cat in which a similar lesion had been produced, except that the catalepsy was much less marked than that obtained by these observers with a dose of 25 mg. per kilogram.

LABYRINTHECTOMIZED CATS

Two cats in which bilateral destruction of the labyrinths had been carried out received doses of 17 and 20 mg. of bulbocapnine per kilogram, respectively. These animals had attained good compensation and were able to move about freely and to care for themselves; some excess movement still persisted in both, with occasional excursions of the head, and most movements were of a quick, jerky type. In walking, a wide base and a low center of gravity were exhibited. Within twenty minutes after the injection of the 20 mg. dose of bulbocapnine drowsiness ensued, and there was noticeable alleviation of the animal's shakiness. The excursions of the head were no longer evident, and the movements in general were more economical and purposeful, although the wide base was retained. Complete submissiveness in adopting poses was not obtained, but when undisturbed the animal was perfectly immobile; it finally went to sleep in the characteristic crouching, flexed position. With the 17 mg. dose similar results were effected. The excursions of the head and jerkiness of movement disappeared. The animal did not adopt poses, but profound drowsiness and the flexion attitude were characteristic manifestations of the intoxication. Specific tests of tonus were not applied in these animals, but the greater steadiness may have been due to some increase in muscular tone. As to the general reactivity of these animals to the drug, it will be seen that it does not differ from that of normal animals which received similar doses.

CATS WITH LESIONS IN THE RED NUCLEI

Six cats in which lesions involving the red nuclei had been produced were used in this experiment. Four received dosages of 20, 15, 10 and 5 mg. per kilogram, with intervals of at least twelve days between each dose. One cat received injections of 20, 10 and 5 mg. per kilogram, and another was given a single dose of 20 mg. per kilogram. The effects of each of these will be described. It should be remembered that cats with lesions in the red nuclei show certain disorders of locomotion, including dysmetria, ataxia and circumduction of the limbs; there is also a mild increase in the tonus of the extensor muscles. The righting reactions remain intact, but in some instances there may be forced movements. Observations on the behavior of such animals have been reported (Ingram and Ranson [1b]). Figure 1 shows the location of the lesions in cat 43.

With the 20 mg. doses, all six cats displayed more profound catalepsy than did the normal cats or those which had been subjected to partial decortication or labyrinthectomy. They showed somnolence, adoption of the characteristic flexion posture and moderate acceptance of passively induced positions of the limbs, or

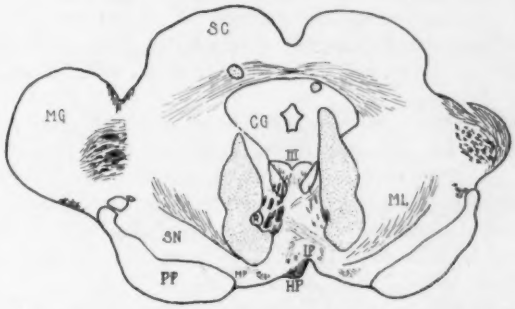

Fig. 1.—Cross-section of the brain stem of cat 43, showing lesions in the red nuclei. *CG* indicates the central gray; *HP*, the habenulopeduncular tract; *IP*, the interpeduncular nucleus; *MG*, the medial geniculate body; *ML*, the medial lemniscus; *MP*, the mamillary peduncle; *PP*, the pes pedunculi; *R*, the red nucleus; *SC*, the superior colliculus; *SN*, the substantia nigra, and *III*, the oculomotor nucleus.

posing. In the supine position the tendency to flex the spinal column was marked, and while the animal was so placed no clearly significant changes in tonus could be demonstrated in the limbs. They were able to support the weight of the body when the forepaws were placed on a high stool in the hanging position, thus manifesting the so-called "active" catalepsy of de Jong. Four of these cats showed forced movements, exemplified by curvature of the spinal column, whereas no such condition had been noticeable before the administration of the drug. This is in accord with the findings of Krisch and Spiegel [3] that certain latent disturbances in tonus may be made evident as postural anomalies under the influence of bulbocapnine.

Four cats of this series received injections of 15 mg.; within from ten to twenty minutes they showed good catalepsy with all the concomitant signs.

3. Krisch, H., and Spiegel, E. A.: Sichtbarmachung latenter, experimentell hervorgerufener Tonusstörungen mit Bulbocapnin, Ztschr. f. d. ges. Neurol. u. Psychiat. **122**:535, 1929.

Induced poses were rather readily assumed, in spite of some resistance to passive movement. When the animals were supine the reactions were the same as with 20 mg. of the drug, and there was good response to hanging. Visible vegetative disturbances were so slight as to be almost overlooked, but it was obvious that animals of this type are much more susceptible to bulbocapnine intoxication, at least where motor manifestations are concerned, than are normal cats which receive similar doses.

Similar symptoms were displayed by five animals after the injection of 10 mg. of bulbocapnine per kilogram, although the catalepsy was not so marked or so prolonged. There were somnolence, a tendency to flexion and posing. All the usual signs except sympathetic disturbances were present; the differences in the effects of larger doses rested in their degree and duration. There were no significant changes in the muscle tonus of the limbs, so far as one could determine from the *Stütz* reflex and resistance to passive flexion.

The same five cats were given 5 mg. of bulbocapnine per kilogram from ten to fourteen days later. Even with such small doses there was considerable evidence of intoxication; in fact, one of these animals showed very fair catalepsy, judging by the usual criteria. The other four did not attain such a profound involvement but displayed marked inertia which bordered on catalepsy and which was much more pronounced than that shown by normal cats which were given similar quantities of bulbocapnine. Here, again, no definite changes in tonus were evident.

In contrast with the cats previously mentioned and with normal animals, those with lesions in the mesencephalic tegmentum obviously possessed a singular susceptibility to the drug, so far as the motor components of the catalepsy were concerned. This did not extend to the vegetative apparatus, however, and no significant changes in the tonus of the musculature of the limbs were detectable, so far as the *Stütz* reaction to stimulation or to passive flexion was concerned, with the animal in the supine or dorsal position. It should be mentioned that two of these cats showed transient symptoms of catalepsy for two days following the operation. These showed no more profound reaction to bulbocapnine than did the others; indeed, the animal which was most affected by the 5 mg. dose had not shown any signs of catalepsy previously.

CATS PREVIOUSLY SHOWING CATALEPSY AS A RESULT OF RETROMAMILLARY LESIONS

In this group there were eight cats in which lesions had been placed in the rostral part of the midbrain and the caudal part of the diencephalon, with resulting tendencies to sleep and catalepsy, as has been outlined earlier in this article. At the time of the first treatment with bulbocapnine all but one had recovered from the catalepsy which had been evident before, although they still showed abnormalities of behavior. Since there were differences in the duration and depth of the catalepsy in the animals and since some of them presented special idiosyncrasies, each animal will be accorded brief consideration. A detailed study of the behavior of these cataleptic cats and of the location and extent of the lesions as determined by microscopic study of their brains will be presented in another paper. Figure 2 shows the location of the lesions in cat 15.

Cat 3.—This animal was operated on on Sept. 9, 1932. The day following the operation there were marked somnolence and indisposition to move about. The body-righting reflexes were intact. Certain abnormal poses could be induced and were maintained for a time. After considerable handling the animal would

arouse from its lethargy and give some signs of resentment. There was a tendency to unusual flexion of the limbs when at rest. When the animal was supine there was evident increase in the tonus of the extensors. The catalepsy in this instance was not especially profound, and after three days it was difficult to induce abnormal poses. The general stupidity and lethargy persisted, however, and for many weeks feeding by tube was necessary; even as long as six months after the operation special effort was necessary to induce the animal to take food. The general behavior during the period of recovery was distinctly abnormal and possibly psychopathic. Periods of rest in odd positions alternated with periods in which the animal moved about its cage emitting peculiar hoarse cries which did not appear to be associated with hunger but which may possibly have been occasioned by gastro-intestinal discomfort, for the stools at one time were found to contain blood.

On November 22, the cat's behavior was at times normal. It could not be induced to pose and was timid and occasionally irritable. Vestiges of increased tonus at this time were detectable only in the forelimbs. At this time 20 mg. of bulbocapnine was administered subcutaneously, and its effects were manifested

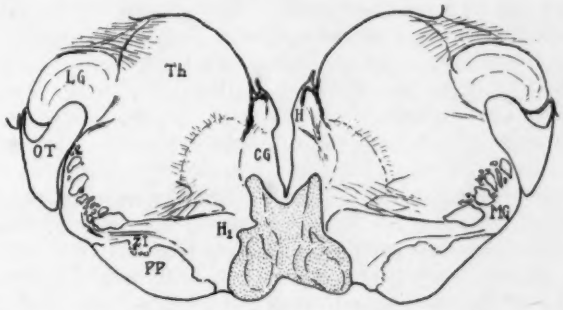

Fig. 2.—Cross-section of the brain stem of cat 15, showing retromamillary lesions. *CG* indicates the central gray; *H*, the habenula; H_1 the H_1 field of Forel; *LG*, the lateral geniculate body; *MG*, the medial geniculate body; *OT*, the optic tract; *PP*, the pes pedunculi; *Th*, the thalamus, and *ZI*, the zona incerta.

shortly by profuse salivation, vomiting, assumption of the flexion position and willingness to adopt poses, including one in which the forelimbs were flexed and the hindlimbs extended in a sort of caricature of an attitude of prayer. There were no significant changes in the tonus of the extensor muscles, so far as could be determined by the usual tests. All in all, a condition of profound catalepsy was induced which exceeded any as yet seen in normal animals or animals which have been operated on.

Fifteen days later, 15 mg. per kilogram was injected. Again there was a profound vegetative upset, resulting in repeated attacks of nausea. As this passed off a rather good state of catalepsy developed, which presented the usual characteristics. After another interval, this time of three weeks, a 10 mg. dose was given; this was followed by the onset of deep catalepsy. There were marked tolerance of abnormal poses and considerable resistance to manipulation of the forelimbs; the flexion tendency was strong, and flexion of the spine was especially evident when the animal was supine. At this time the forelimbs were plastic and showed increased tonus, as was indicated by considerable resistance to passive movement; the hindlimbs showed chiefly an increased tone of the

flexors. The body-righting reflexes were present, and the hanging reaction was good. A singular feature, not noted with larger doses, was that the head was now held twisted slightly to the right, whereas when the animal was not under the influence of the drug there was a slight tendency to forced movement of the head to the left.

The last dose, 5 mg. per kilogram, was administered eleven days later. Again a good state of catalepsy resulted, which was essentially similar to that caused by the 10 mg. dose, except that now the plasticity of the hindlimbs exceeded that of the forelimbs when the cat was on its back. The reversal of forced movement of the head was evident again.

In summary, doses of from 5 to 20 mg. of bulbocapnine per kilogram of body weight induced a cataleptic condition which was much more profound than that induced by such doses in normal cats.

Cat 4.—This animal was operated on on September 12. On recovery from the anesthetic there were considerable lethargy and somnolence, together with rather marked catalepsy. Passively induced positions were retained, and peculiar attitudes were occasionally assumed spontaneously. There were slightly increased tonus of the extensors and rather good plasticity of the limbs when the animal was supine. During the succeeding weeks there was gradual improvement. For the next month, however, the cataleptic tendencies were evident most of the time, but periods of spontaneous activity were occasionally observed. The animal could be induced to take food only by considerable coaxing, and occasionally feeding by tube was necessary. By November, however, recovery was so far advanced that feeding was spontaneous, and there were no particular evidences of catalepsy. However, the animal was distinctly different from normal cats. It would sit for long periods with a vacuous, stupid expression and during other periods would circle restlessly in its cage. There was a marked forced movement which was characterized by curving of the spinal column with concavity to the left, and when walking the cat always circled strongly to the left. The tonus of the muscles of the limbs was practically normal.

On November 21, an injection of 35 mg. of bulbocapnine per kilogram was administered. Shortly afterward there were profuse salivation and stupor. The animal sat on its haunches, with its head thrown back so that its nose pointed to the ceiling, and considerable resistance was offered when attempts were made to push the head forward. As the drug continued to take effect profound catalepsy ensued, and the cat could be posed in various positions, including the attitude of prayer as well as the hanging position. There was practically no change in tonus in the limbs when the cat was supine. When the animal lay on its side a persistent flexion of the limbs was conspicuous. At this time it was observed that the head was now turned slightly to the right. Similar results were obtained after the injection of 15 mg. per kilogram, two weeks later. Deep catalepsy again appeared, and again it was noted that the forced movement, which before the administration of the drug was normally characterized by curving of the spine to the left, was now modified so that the head was turned slightly to the right. The effects of the drug were not so prolonged in this instance.

The injection was repeated with a 10 mg. dose after an interval of about three weeks. All the effects previously noted were again in evidence. When the animal was supine no marked rigidity of the limbs or enhanced *Stütz* reaction was detectable, but there was some slight plasticity of the whole body and of the limbs to varying degrees of flexion and extension. Ten days later a dose of 5 mg. per kilogram was followed by a more severe vegetative upset than had hitherto been noticed, and there was some vomiting. The catalepsy was again

marked; poses and hanging were well maintained; there were reversal of the forced movement and marked stupor and tendency to flexion. When the animal was supine there were some resistance to passive movement of the limbs and fair plasticity.

To summarize, the cataleptic effects of the 35 mg. dose of bulbocapnine were reproduced in cat 4 by doses of 15, 10 and 5 mg. per kilogram. There was much greater susceptibility to the drug than is found in normal animals or in animals with lesions in other regions of the nervous system.

CAT 9.—This animal was operated on on September 23. During the days immediately following the operation it showed a pronounced tendency to sleep and marked irritability when aroused. There was forced curvature of the spine, which was concave, to the right. There was fair resistance to stimulation of the *Stütz* reflex, and one obtained an impression of considerable spasticity of the hindlimbs. By October 3 the irritable phase had passed off, and the cat could be induced to pose in the aforementioned attitude of prayer. At this time some slight general increase in the tonus of the limbs was noticeable. Forced feeding was necessary. Figure 3, *A* to *D*, portrays the condition of this cat on October 14.

On November 22 there were still some signs of catalepsy, with fair acceptance and maintenance of various induced poses. The curvature to the right persisted, and there was also a tendency to ventral flexion of the trunk. When the cat was lifted from the ground the observer received an impression of great rigidity of the muscles of the neck, trunk and limbs; indeed, when the trunk was supported by the hand under the belly the limbs showed remarkable extension. However, when the animal was in the supine position, a positive *Stütz* reaction in response to stimulation was not marked. There was great disinclination to move about, and usually the animal stood in one spot for long periods, only occasionally shifting its position. Under such circumstances peculiar attitudes were frequently assumed spontaneously. Sometimes the animal would lie on its side with the head raised and the limbs curved up clear of the floor. It showed a consistent degree of awareness, noticing moving objects and occasionally greeting the observer with cries when he entered the room. At this time 25 mg. of bulbocapnine per kilogram was administered. In a short time there was excessive salivation, followed by vomiting, and thereafter a statuesque pose was assumed in which the head was held up, the forelimbs were partly extended and the hindlimbs were flexed at the hip and knee but extended at the ankle so that the trunk was supported clear of the floor. There was considerable waxy flexibility, and various poses could be passively induced, including the attitude of prayer. When the animal was placed in the supine position there was strong flexion of the trunk, which could be modified by manipulation; there was no *Stütz* reaction, but some plasticity of the forelimbs and great resistance to passive extension of the hindlimbs were noted. There was profound somnolence, but painful stimuli evoked crying and hissing. The effects were of unusually long duration and were more profound than those seen in any other cat, either normal or operated on. Figure 3, *E* to *H*, shows some of the attitudes which this cat could be induced to take after the administration of 25 mg. of bulbocapnine.

Similar results were obtained two weeks later, when 15 mg. per kilogram was injected. The tendency to the flexion position was striking, and immobility was marked. There was good acceptance of passively induced postures, and the cat could support itself in the hanging position. Recovery was greatly delayed.

After another interval of three weeks, 10 mg. per kilogram was given. Vomiting and manifest discomfort followed, and the usual cataleptic syndrome developed.

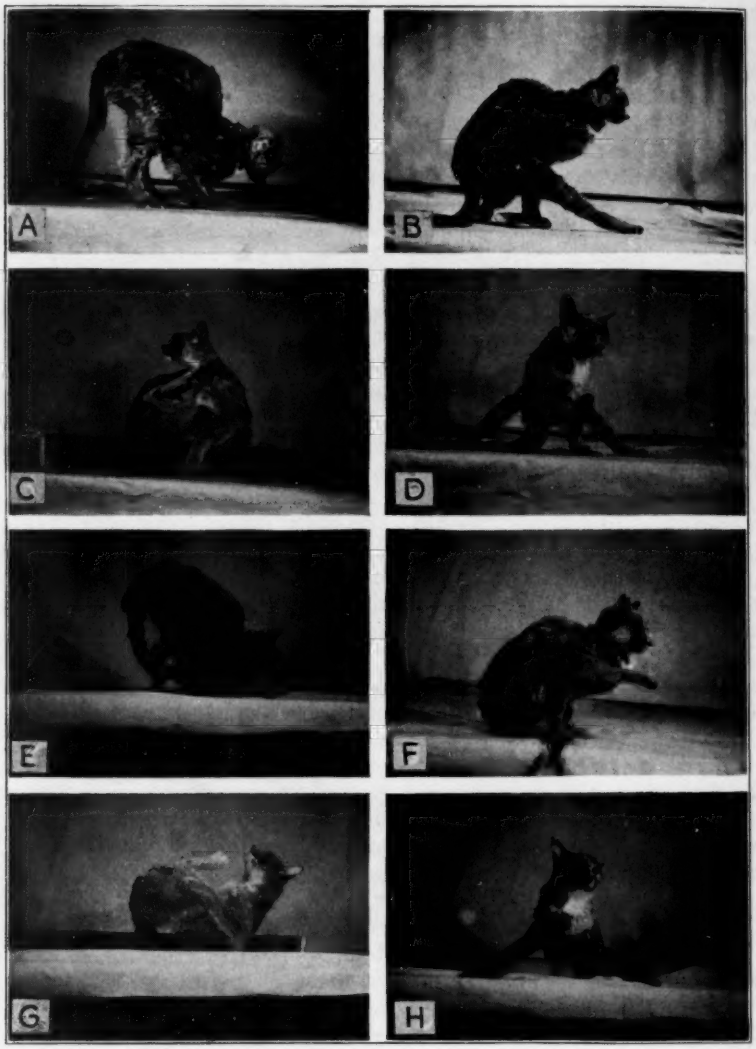

Fig. 3.—*A* to *D*, inclusive: photographs of cataleptic cat 9 in various passively induced poses, taken October 14, six weeks before the administration of bulbocapnine. *E* to *H*, inclusive: photographs of cat 9 in various passively induced poses while under the influence of bulbocapnine (25 mg. per kilogram of body weight); taken November 22.

When the animal was supine there was good plasticity but practically no accentuation of the *Stütz* reaction. There was considerable resistance to manipulation.

The final dose of 5 mg. per kilogram again elicited signs of vegetative upset, which was marked by urination, defecation and vomiting. This was followed by profound immobility and willingness to retain various poses. When the animal was placed on its back its behavior was similar to that which had been observed after the administration of larger amounts of the drug. Again there was a state of deep catalepsy, which was of long duration.

This cat, the behavior of which was decidedly abnormal in the chronic stage following the operation and which showed decided lack of motor initiative, proved more susceptible to the effects of bulbocapnine than any other cat, either normal or operated on, in our series.

CAT 11.—This animal was operated on on September 29. Rather pronounced somnolence was observed for several days following the operation, during which time the animal could be induced to retain various poses, and there was definitely increased tonus of the extensor muscles. By the fifth day there was more spontaneous movement, and there were indications that considerable portions of the red nuclei had been destroyed, since the gait was rather dysmetric and the pupils were somewhat dilated. For some time thereafter the animal's behavior was marked by general stupidity, but although the cat could be handled with ease no real catalepsy was evident, and the muscle tonus approached normal. There was a slight indication of forced movement to the right.

On November 29, when the initial dose of 20 mg. of bulbocapnine per kilogram of body weight was given, the cat appeared normal except for the symptoms of involvement of the red nucleus. After the administration of the drug the cataleptic manifestations developed rather slowly, without vegetative symptoms, to an unusual depth. All the typical motor characteristics were observed, and there was a tendency to curvature to the left. Similar results were obtained on December 12 after the injection of 13 mg. per kilogram. The catalepsy was striking, and when the animal was placed on its back there appeared to be a sort of waxy rigidity of the limbs, which was marked by resistance to passive movement and by a fair degree of plasticity. This was also true after a dose of 10 mg. per kilogram, given on January 5.

In general, it may be said that cat 11, with doses of 10, 13 and 20 mg. of bulbocapnine, showed a depth of catalepsy which would be exceedingly unusual in normal animals.

CAT 12.—This animal was operated on on October 27. It exhibited a fair degree of catalepsy for a period of about four days following the operation. After this there was no evidence of true catalepsy, except that the cat was unusually quiet and occasionally would strike an odd attitude. Changes in tonus were slight and of short duration. In walking there appeared to be slight hypermetria of the right limbs, and there was a tendency to turn to the right.

On November 29, after the injection of 20 mg. of bulbocapnine per kilogram, vomiting and urination ensued, followed by indications of motor catalepsy. The catalepsy became marked, and various poses, including hanging, were retained. There was slightly increased muscle tonus, which chiefly affected the forelimbs. The forced movement was reversed, so that the head was now turned to the left. No other injection was given until Jan. 23, 1933, because of the development of an infection of the inner ear, with resulting vestibular disturbances. The administration of 5 mg. per kilogram at this time caused vegetative upset and

inertia, which were followed by catalepsy of a rather low degree. Induced poses were accepted but were not retained as well as usual. A marked forced movement to the left developed. The response to small doses of the drug was not so marked in this cat as in the other animals of this series.

CAT 15.—This animal was operated on on November 3. Following the operation there were drowsiness, inertia and a fair degree of catalepsy, but practically no increase in tonus. Passively induced poses, including the attitude of praying, were fairly well maintained. This condition persisted for more than a week, but by November 16 the animal had recovered rather well. However, the behavior never returned completely to normal. The cat was quiet, often assumed odd attitudes and took food only with considerable coaxing.

On December 6, 20 mg. of bulbocapnine was injected, and shortly afterward the effects were made evident by the occurrence of salivation and vomiting. A statuesque squatting posture was taken, and certain poses, including hanging, could be passively induced. When the animal was supine the trunk was flexed strongly, but no change in tonus in the limbs was observed. The animal's stupidity and inertia were greatly intensified.

Fifteen milligrams per kilogram was given about a week later. The results were similar in every way to those obtained with 20 mg. There was good ability to retain the hanging position.

The behavior of this cat under normal circumstances was always peculiar. At this time forced feeding was necessary, and although the animal was alert and active at times, there were intervals in which fixed postures were maintained. On Jan. 5, 1933, 10 mg. of bulbocapnine per kilogram was given, and a fair degree of catalepsy resulted. A good attitude of praying could be induced, but there was some difficulty in imposing a supine position. There was a good hanging reaction. A 5 mg. dose, given on January 23, produced a somewhat more profound catalepsy. The supine position was accepted, and while the animal was thus placed various degrees of flexion of the trunk could be imposed. There was no increase in the tonus of the limbs, but contact with the pads of the feet called forth sudden, jerking movements. The hanging position was retained for fifteen minutes.

It was evident that cat 15, while not so susceptible to bulbocapnine as some other animals of this series, was more easily affected by the drug than are normal animals.

CAT 18.—This cat was operated on on November 10. It showed considerable involvement of the motor apparatus, which lasted for several days. There was difficulty in standing and in righting the body when it was placed so that the right side was down. The spinal column was curved, with the concavity to the right. There was markedly increased tonus of the extensor muscles, which was especially evident in the right limbs. Along with this there were profound somnolence and rather good catalepsy; various poses could be induced within the limits imposed by the forced movement. After a week the cat began to wake up and show better coordination and control of movements. The cataleptic tendencies persisted for a short period thereafter, but considerable spontaneous movement was soon manifest, and there was some diminution of the muscular rigidity. The gait remained abnormal, being characterized by hesitation, slowness and awkwardness; the forced movement to the right persisted.

On December 6, 20 mg. of bulbocapnine per kilogram was injected. Within a very short time there was excessive salivation, and the typical flexion attitude was assumed. The forced movement was reversed, so that the head and fore

part of the body were curved strongly to the left. Various poses could be induced, and when the animal was supine the flexion of the spine became especially well marked. There was little increase in the tonus of the muscles of the limbs, and such increase as there was affected chiefly the flexors. The hanging reaction was rather good. Profound and persistent somnolence was an additional symptom.

Fifteen milligrams per kilogram had very similar effects, producing good catalepsy. Subsequent doses of 5 and 10 mg. per kilogram also produced good catalepsy, but this was less deep. In each instance it was prolonged beyond the usual time limit.

CAT 21.—This animal was operated on on January 10. Following the operation good catalepsy developed which lasted for about ten days. While the animal was in the cataleptic state all the typical manifestations were observed: There was a tendency to forced movement, with curvature to the left; the hindlimbs showed definitely increased extensor tonus, and there was good plasticity of all the limbs. As recovery supervened and the animal became more active, the tonus of the limbs approached normal, but the forced movement persisted. From time to time there were slight relapses to a state resembling catalepsy.

One injection of 5 mg. per kilogram was given to this animal on March 9. This was followed by salivation and vomiting, the assumption of an odd, semi-flexed position and drowsiness. Passively induced poses were readily assumed, including the attitude of prayer and the hanging attitude. When the animal was supine the tendency to flex the spine was marked, but there was no significant change in the *Stütz* reaction. When at rest the cat was immobile and statuesque; it was apparently unable to move a limb, although the proper position of the body was maintained. The forced movement was hardly affected; although there was a slight tendency to turn the head to the right, the trunk remained curved to the left. All in all, it was evident that this animal reacted much more readily to small doses of bulbocapnine than do normal cats.

EFFECT OF REPEATED DOSES

Although long intervals between doses of bulbocapnine were allowed in the animals which were subjected to repeated injections, it was thought that objection might be made on the grounds of a possible cumulative effect or increased susceptibility to the drug. Therefore, in a series of four normal animals successive doses of bulbocapnine were given over a period which allowed shorter intervals of recovery than in the previously described experiments. Two cats were each given two doses of 20 mg. per kilogram at intervals of three days. These showed rather good average catalepsy in response to each injection, although this was not so profound as that seen in cats with lesions of the brain stem. A week later these animals were given 15 mg. per kilogram, and again rather good catalepsy ensued. At this time these cats were found to be in rather poor physical condition, and one was killed. The other was given injections of 10 mg. per kilogram after another week had elapsed; this time the catalepsy was less profound. A week later 5 mg. per kilogram produced still milder effects. Two cats were given 15, 10 and 5 mg. per kilogram. In one of these the larger dose caused moderate catalepsy of rather short duration. The 10 mg. dose also caused loss of motor initiative of a similar degree. Five milligrams per kilogram had much less effect, and no true catalepsy resulted. The fourth cat showed a very mild degree of catalepsy after receiving 15 mg. per kilogram; with 10 mg. per kilogram there was considerable inertia but practically no active catalepsy; 5 mg. per kilogram produced nothing more than a slight diminution of motor activity and a tendency to drowsiness.

The results of these experiments led us to believe that there is no cumulative effect of successive doses when several days are allowed to elapse between injections. In no case were the effects so profound as those observed in cats with lesions of the brain stem. It should be pointed out, however, that Pero [4] found cats to be more susceptible to bulbocapnine than dogs and rabbits. He observed that repeated daily doses of 20 mg. produced the same degree of catalepsy in cats, while in dogs and rabbits it was necessary to increase the dose in order to reinduce catalepsy of the same depth.

As a further check on this point, cats 3 and 4, which, as has already been described, had previously shown catalepsy as a result of retromamillary lesions, were each given 5 mg. of bulbocapnine per kilogram on June 28, 1933. A period of five and a half months had elapsed since they had last received bulbocapnine. Both showed profound catalepsy, with remarkable acceptance of induced poses and hanging reactions. The effects were much more profound than in normal cats which received larger doses. It may also be pointed out that cat 21, which received only a single administration of 5 mg. per kilogram, showed a marked reaction to bulbocapnine, as has already been described.

These experiments, in our opinion, rule out the possibility that successive injections of the drug could have had anything to do with the profundity of the catalepsy produced by the small quantities of bulbocapnine used in the animals which had been operated on.

COMMENT

In recent years the experiments of de Jong and his co-workers have focused considerable attention on the physiologic effects of bulbocapnine when administered to various types of animals and to man. Although this alkaloid has long been known to pharmacologists, the interest lately aroused has been due chiefly to the supposed similarities between bulbocapnine intoxication and the catatonic phenomena observed in certain cases of dementia praecox. This comparison has been carried to great length by de Jong and others (Baruk and de Jong [5] and Krause and de Jong [6]), who postulated a direct action of the drug on the cortex cerebri and who brought forward data from their experiments as evidence in support of the theory of the toxic origin of catatonia. In this they have been upheld to a certain extent by Spiegel,[7] who believes that

4. Pero, C.: Alterazioni istologiche del sistema nervoso centrale nell'intossicazione sperimentale da bulbocapnina, Schizofrenie **1**:3, 1932.

5. Baruk, H., and de Jong, H.: Etudes sur la catatonie expérimentale; l'épreuve de la bulbocapnine chez divers animaux avec et sans néopallium, Rev. neurol. **2**:532, 1929; L'épreuve de la bulbocapnine chez les singes. Comparison des stades de l'intoxication bulbocapnique avec les aspects de la catatonie humaine, ibid. **2**:541, 1929; La catatonie expérimentale par la bulbocapnine, Paris, Masson & Cie, 1930.

6. Krause, F., and de Jong, H.: Ueber die Lokalisation einiger motorischer Erscheinungen bei der Bulbocapnine-Katatonie, Ztschr. f. d. ges. Neurol. u. Psychiat. **133**:754, 1931.

7. Spiegel, E. A.: Zur Pharmakologie der zentralen Tonusinnervation, Deutsche Ztschr. f. Nervenh. **124**:105, 1932.

at least a certain group of stuporous conditions is due to toxic inhibition. He pointed out that the stupor caused by bulbocapnine may be broken through by the same means which are effective in the catatonic state in man. In this regard, confirmative results have been presented by Paterson and Richter [8] and Buchman and Richter.[9]

A general review of the extensive literature related to bulbocapnine is not required here in view of its detailed consideration in the recently published work of Ferraro and Barrera.[10] These investigators presented the results of a long series of experiments on cats and monkeys, in which the effects of bulbocapnine were studied in animals which had been subjected to various operative procedures, such as partial decortication, decortication plus removal of the striatum, decerebellation, a lesion of the mesencephalon severing the decussation of Forel, hemisection of the spinal cord, section of spinal nerve roots and sympathectomy. The results of these experiments indicated that cataleptic manifestations could be induced in the chronic as well as in the acute stages following all these operations, possibly even affecting deafferented or deefferented limbs, and the authors believed that they had successfully confuted the ideas of those who think that the presence of the cortex is necessary for the occurrence of bulbocapnine catalepsy. It was held that no psychic component was detectable and that therefore the identification of catalepsy due to bulbocapnine with human catatonia was not justified. This, of course, controverts the suggestions of de Jong and his group and the earlier work of Girndt and Schaltenbrand.[11] Schaltenbrand, however, has more recently found that the symptoms of bulbocapnine intoxication in striatal cats are not different from those occurring in normal animals.[2] Sager [12] concurred with this and suggested that bulbocapnine has an inhibitory action on the diencephalomesencephalic centers as well as on the cortex.

The results of the present work in general confirm the findings of Ferraro and Barrera. The occurrence of catalepsy due to bulbocapnine

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8. Paterson, A. S., and Richter, C. P.: Action of Scopolamine and Carbon Dioxide on Catalepsy Produced by Bulbocapnine, Arch. Neurol. & Psychiat. **29:** 231 (Feb.) 1933.

9. Buchman, E. F., and Richter, C. P.: Abolition of Bulbocapnine Catatonia by Cocaine, Arch. Neurol. & Psychiat. **29:**499 (March) 1933.

10. Ferraro, A., and Barrera, S. E.: Experimental Catalepsy (The Action of Bulbocapnine in Cats and Monkeys with Various Experimental Lesions of the Nervous System), Monograph, Dept. of Neuropathology, New York State Psychiatric Institute and Hospital, New York, Utica, N. Y., State Hospitals Press, 1932.

11. Girndt, O., and Schaltenbrand, G.: Die Wirkung des Bulbocapnins auf Thalamuskatzen, Arch. f. d. ges. Physiol. **209:**333, 1925.

12. Sager, O.: Experimentelle Untersuchungen über die Bulbocapninstarre (Zugleich ein Beitrag zum Mechanismus der Katalepsie), Ztschr. f. d. ges. exper. Med. **81:**543, 1932.

following lesions of the motor area of the cortex, lesions of the mesencephalon and sympathectomy, as well as labyrinthectomy, has been observed. The additional observation has been made that after bilateral lesions have been produced in the region of the red nuclei the susceptibility to small doses of bulbocapnine is increased. This increased susceptibility is more evident in cats which, as the result of damage to the retromamillary region, have displayed postoperative catalepsy as previously described. It is a rather anomalous situation in which the removal of part of the central nervous system renders the latter more susceptible to the effects of a drug, and one is at first inclined to account for it on the basis of a weakened condition of the animals. However, all the cats used in these experiments were in excellent general health. A satisfactory explanation of this situation cannot be advanced at the present time. Any suggestion which might be made must be purely speculative and could not be supported adequately, nor can suitable evidence be presented as to whether bulbocapnine is excitatory or inhibitory in its action. It can scarcely be without significance, however, that a lesion which causes postoperative catalepsy leaves the animal especially susceptible to the action of bulbocapnine after the catalepsy following a retromamillary lesion has disappeared.

There are a number of minor points of interest in connection with our experiments which may be mentioned briefly. In the case of the bilaterally labyrinthectomized animals bulbocapnine appeared to cause a diminution of the excursions of the head and to exercise a general steadying effect on all the movements of the body. Somewhat similar results have been noted clinically by various authorities in cases of cerebellar injury, paralysis agitans, Benedikt's syndrome and Huntington's chorea and have been ascribed to general inhibition, sedation or generally increased muscular tonus. It seems likely to us that in our cats there may have been a combination of all these factors.

Various investigators have remarked on the vegetative phenomena which are frequent precursors of the onset of catalepsy due to bulbocapnine. In our experiments on normal cats such effects as salivation, vomiting, urination, defecation and the like were seen occasionally but did not occur consistently. Usually the smaller doses were most effective in evoking such symptoms. It is striking, however, that in animals with lesions of the brain stem, especially in the retromamillary area, these effects were common. This may be considered of some significance when one recalls that these vegetative symptoms are of a parasympathetic nature and that the lesions in these cats were so placed as to involve, very likely, fibers descending from the nuclei of the hypothalamus.

Another interesting observation was that bulbocapnine frequently reversed the direction of a forced movement which remained as a residual symptom after recovery from a lesion of the brain. In this connection the work of Krisch and Spiegel [3] may be recalled. These authors found that bulbocapnine was capable of calling forth latent changes in tone in animals affected with injury to a hemisphere which involved the motor area and the corpus striatum. These changes in tone were made manifest by such postural anomalies as torticollis. It is, of course, difficult to draw a parallel between these results and ours. In our cases the striatum could have been involved only indirectly, and it is possible that the spontaneous forced movement was due to involvement of the supravestibular connections located in the midbrain. This pleurothotonus may be produced by diminished tonus of the muscles of the neck and trunk of one side of the body, but any attempt to account for its reversal by bulbocapnine, on the basis of the action of the latter on either the central or the peripheral areas, can be but pure speculation.

So far as the effect of the drug on the tonus of the hindlimbs is concerned, the results in our cases were rather varied. In general, it caused no significant change in the *Stütz* reaction in the animals which had been operated on. That great variations in the effect of bulbocapnine on tonus may be found in normal animals is indicated by our own observations and by noting the conflicting reports and interpretations which are to be found in the literature. However, there was a definite increase in the tonus of the flexor muscles of the neck, trunk and abdominal wall. This was brought out when the animals were placed in the supine position, where the high tension of the abdominal muscles was a contributing factor in bringing the trunk into an attitude of flexion. This tendency to ventroflexion of the spine was a marked feature of both postoperative catalepsy and catalepsy due to bulbocapnine.

In conclusion, it may be said that while it is perhaps improper to draw a direct parallel between the catalepsy caused by bulbocapnine intoxication and human catatonia, the fact remains that the phenomena observed in these conditions are similar. There is also great similarity between catalepsy produced by bulbocapnine and the condition resulting from retromamillary lesions. It is especially striking to us that animals which had exhibited a form of catalepsy due to lesions involving the upper part of the brain stem should show special susceptibility to bulbocapnine. As yet it is difficult to draw specific conclusions from this fact, but it is to be hoped that it may some time aid in further elucidating the complex physiology of this portion of the central nervous system.

SUMMARY

1. The effects of various doses of bulbocapnine were observed in normal animals and in animals subjected to such operative procedures

as partial decortication, sympathectomy and labyrinthectomy. The action of the drug was essentially the same after these operations as in normal animals. In labyrinthectomized cats the injection decreased the tremor and resulted in increased economy of movement.

2. Varying small doses of the drug were tried in animals with lesions of the mesencephalon involving the red nuclei. These showed much greater susceptibility to bulbocapnine than did normal animals, and good catalepsy could be produced in them by amounts which would ordinarily be inadequate.

3. The catalepsy resulting from bulbocapnine intoxication was found to be similar to that produced by lesions in the retromamillary region of the brain stem. Cats which had previously displayed catalepsy due to retromamillary lesions were found to be exceedingly susceptible to the effects of bulbocapnine, and even after recovery from the effects of the operation a very small dose of the drug was capable of reinducing a cataleptic state of great depth.

BASOPHILIC SYNDROME OF THE PITUITARY

PITUITARY BASOPHILISM (CUSHING)

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During the past decade the hormonal physiology of the pituitary gland has been enriched by conclusive evidence from the research laboratory. These studies have more than confirmed the results of numerous clinical observations, beginning with Pierre Marie's description of acromegaly in 1892. However, the clinical story has not yet been fully told, for in the past year Cushing [1] described a new syndrome which he called "pituitary basophilism." This is a striking clinical picture associated in most instances with a verified adenoma of the basophil cells of the anterior lobe. He has so convincingly established this entity that, though it may be elaborated further, it will remain as the outstanding Cushing syndrome.

I present a number of cases with the desire to confirm Cushing's observations and to show that the basophilic symptoms and signs may predominate in other pituitary syndromes, not all necessarily associated with an adenoma of the basophil cells of the anterior lobe.

Ten of the cases reported by Cushing were fatal, and six of these presented verified basophil adenomas. Of the other four, two showed undifferentiated adenomas of the pituitary gland and two were said to present a normal gland (serial sections were not made). The remaining six cases included two in which neither autopsy nor operation was performed; in these the patients presented characteristics formerly considered suprarenal and now thought to be both pituitary (basophil) and suprarenal; in two others the symptoms of the basophilic syndrome were striking and the patients recovered after the application of roentgen therapy to the pituitary gland; the remaining two patients demonstrated the basophilic syndrome in part; one recovered without treatment and the other is still under observation.

The striking uniformity of the clinical signs observed in these cases is noteworthy. The symptoms and signs fall into the following groups: (1) adiposity of the face and trunk, usually sparing the extremities; (2) amenorrhea, or sexual impotence in the male; (3) hypertrichosis

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Read at the Fifty-Ninth Annual Meeting of the American Neurological Association, Washington, D. C., May 9, 1933.

1. Cushing, H.: Pituitary Body, Hypothalamus and Parasympathetic Nervous System, Springfield, Ill., Charles C. Thomas, Publisher, 1932.

of the face and trunk (masculine in type) in females and adolescent males, and possibly the reverse in adult males; (4) dusky or plethoric skin, with purplish lineae atrophicae, acrocyanosis, cutis marmorata and purpura-like ecchymoses; (5) vascular hypertension; (6) a tendency to polycythemia and polynucleosis; (7) osteoporosis, with softening of the bones of the skeleton and kyphosis; (8) headache, pain in the back, asthenia and fatigability; (9) hyperglycemia and albuminuria; (10) intracranial signs, with exophthalmos, diplopia, papilledema, dimness of vision, polyphagia, polydipsia and polyuria.

All the cases reported showed a striking uniformity of symptomatology. The syndrome was most frequent in young persons of short stature and of an average age of 18 years. In the fatal cases the average duration of the illness to death was about five years.[2] The three patients who recovered, one of them spontaneously, are still under observation.

Since the description of the basophilic syndrome, shortly over a year ago, it has been brought more and more to my attention that numerous causes of virilism are not altogether typical of the aforementioned group. However, whether they present pure suprarenal or combined suprarenal and pituitary syndromes, these cases are of special interest in an attempt at evaluating the factors that contribute toward an excessive growth of hair. Some of the patients, in whom hypertrichosis is a readily recognizable sign, have on further study been found to present a sufficient number of the signs of the basophilic syndrome to allow them to be placed unequivocally in this broader group, leaving it to the future to settle the question of the presence or absence of a basophilic adenoma.

The following reports of cases are in some instances brief because of lack of cooperation; as some of the patients were seen at the clinic it was difficult to obtain complete laboratory studies.

REPORT OF CASES

CASE 1.—*Gradual development of hypertrichosis, abdominal obesity, kyphosis, osteoporosis of the skull, purplish striae, polycythemia, polynucleosis, exophthalmos and increased blood pressure.*

History.—R. S., single, aged 22, came to the Vanderbilt Clinic in 1923. An excessive growth of facial hair, which had first appeared at 16 years, had been removed by roentgen rays at 20. In the clinic for cutaneous diseases a somewhat myxedematous appearance of the skin with telangiectases was noted, and the condition was diagnosed poikiloderma atrophica vasculare. In April, 1929, she was referred to the Neuro-Endocrine Clinic by Dr. B. Kesten, who suspected a tumor of the pituitary gland. At that time it was recorded that the menses

2. Since this paper was written Cushing has described a case which remained under observation for fourteen years, ultimately ending in death. The patient presented "waves of basophilism," and at autopsy a basophil adenoma was found (Cushing, H.: Dyspituitarism: Twenty Years Later, Arch. Int. Med. **51**:487 [April] 1933).

had begun at 12; they were of a twenty-eight day cycle and seven days in duration, and there had been no cessation during their course.

Examination.—There were: kyphosis; torso-leg ratio, 45:78; exophthalmos; marked telangiectasis of the face and chest; mottling of the skin of the extremities; purplish lineae atrophicae on the abdomen and thighs, and masculine pubic hair. The visual fields showed slight contraction temporally. The blood pressure was 140 systolic and 90 diastolic, and the pulse rate was 80. The basal metabolic rate was minus 15 per cent. A blood count revealed: hemoglobin, 100 per cent; red cells, 5,130,000; white cells, 10,200; polymorphonuclears, 80 per cent; lymphocytes,

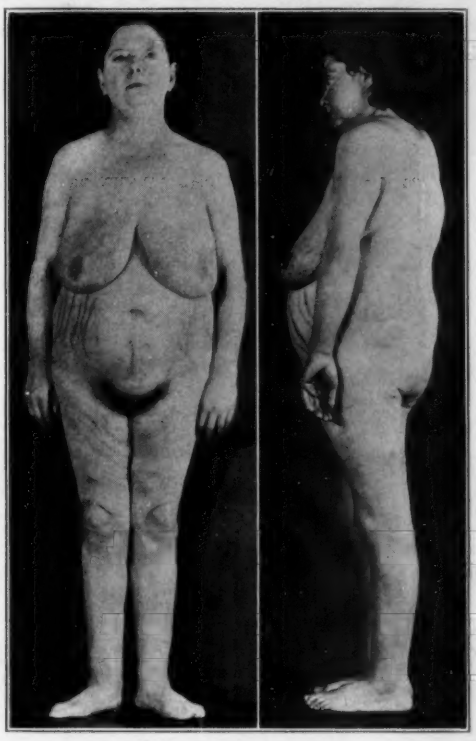

Fig. 1 (case 1).—Patient with typical case of pituitary basophilism. Note the abdominal obesity and the thin legs.

16 per cent; transitional cells, 4 per cent. The blood contained: sugar, 83.3 mg. per hundred cubic centimeters; urea nitrogen, 10.2 mg.; uric acid, 2.6 mg.; calcium, 10.9 mg., and carbon dioxide, 50.4 mg. A roentgenogram of the skull revealed a diffuse decalcification of the skull and dorsum of the sella turcica. No evidence of erosion or tumor was found.

Diagnosis.—It was concluded in conference that the patient had a hypothyroid and hypopituitary condition.

Course.—Treatment with thyroid, pituitary and iodine preparations brought some improvement in alertness, and the patient understood questions better. In March, 1932, Dr. J. M. McKinney noted the syndrome of purplish striae, abdom-

inal obesity, exophthalmos, hypertrichosis, masculine crines and a blood pressure of 150 systolic and 110 diastolic. Ophthalmologic examination showed the fields to be complete, with some constriction of the field for red temporally in both eyes. A roentgenogram of the skull showed the inner table to be hazy owing to

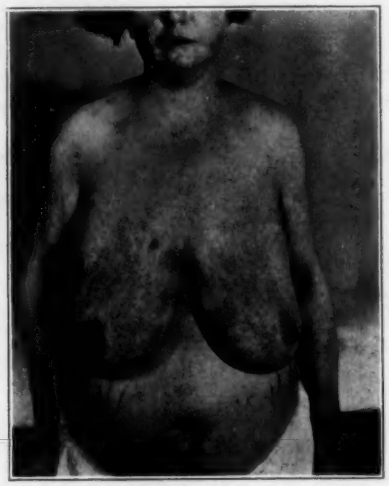

Fig. 2 (case 1).—Purplish striae and purpuric discoloration over the breasts are present.

Fig. 3 (case 1).—Roentgenogram showing generalized atrophy and decalcification of the skull.

the absorption of calcium. The sella turcica was wide open and measured 11 mm. in the anteroposterior diameter, showing a generalized atrophy. A diagnosis of a basophilic adenoma of the pituitary gland was made and roentgen treatment prescribed. After these treatments there was less fatigue, and infections present in the toes disappeared.

Comment.—In this patient there was the characteristic syndrome of pituitary basophilism, lacking only the amenorrhea and the rapid course to death. However, the picture presented is identical with previously reported cases and leads me to believe that there are less rapidly fatal cases of this disease;[2] it also throws light on the uncertain relationship of the menses to the basophil cells of the anterior lobe.

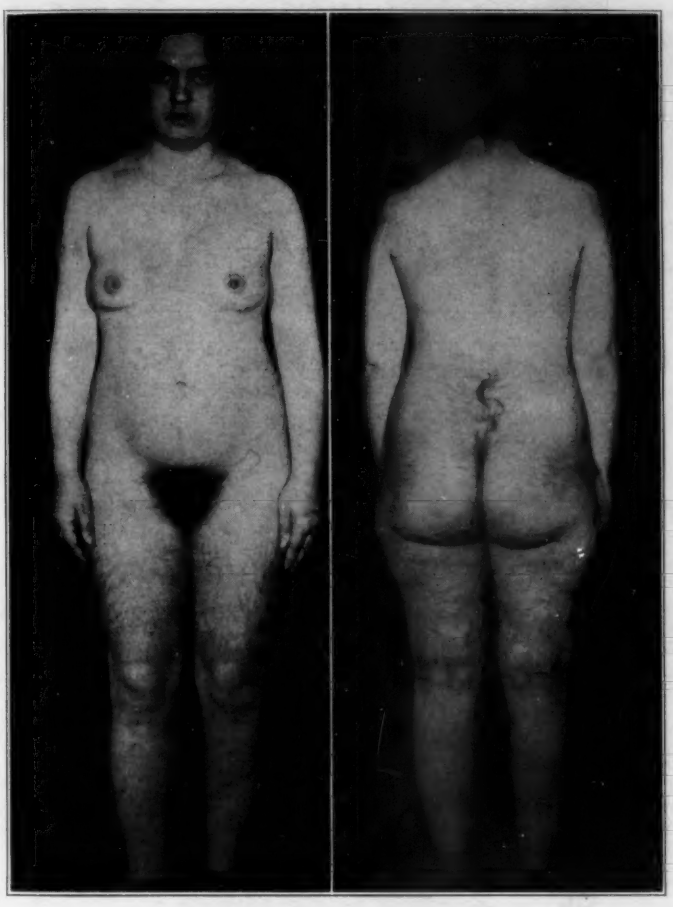

Fig. 4 (case 2).—The sacral hypertrichosis and striae over the popliteal region are striking.

CASE 2.—*Amenorrhea, purplish lineae atrophicae, hypertrichosis of the face and body, rapid gain in weight, low sugar tolerance, osteoporosis of the spine.*

History.—C. R., single, aged 24, a patient of Dr. I. Margaretten, was admitted to the Neurological Institute in November, 1932, complaining of headache, rapid gain in weight and swelling of the face and neck for the past year. In addition there had been an increase in the growth of hair and amenorrhea for the past five

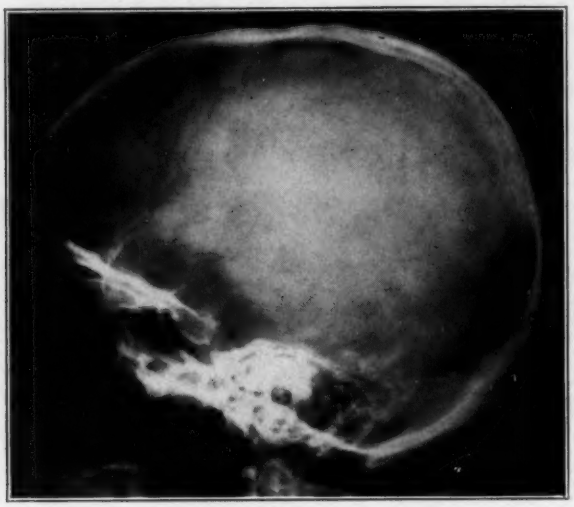

Fig. 5 (case 2).—Roentgenogram of the skull, showing the small sella turcica and a slight atrophy of posterior clinoid processes.

Fig. 6 (case 2).—Roentgenogram of the spine, indicating atrophy of the cancellous portion of the bodies of the dorsal vertebrae.

months. Some diminution of vision had been noted, and an abscess at the tip of the coccyx had failed to heal. The menses began at 14, were always slightly irregular, occurring at an interval of twenty-eight days, and lasted five days. Menstruation occurred twice in May; since July there had been no period.

Examination.—The patient appeared somewhat obese, the adiposity being least in the legs. The skin was dry and doughy and showed cutis marmorata over the deltoid region, breasts and thighs. Over the upper parts of the thighs, hips and knees and in the axillae there were reddish-blue striae (purplish lineae atrophicae). The growth of hair was striking; it was marked on the face, coarse and long over the extremities, of masculine type over the pubes and profuse over the sacral region. The blood pressure was 110 systolic and 80 diastolic; the pulse rate was 90. A laboratory examination of the blood revealed: hemoglobin, 81 per cent; red cells, 4,500,000; white cells, 10,750; polymorphonuclears, 70 per cent; lymphocytes, 26 per cent; urea, 12.3 mg.; sugar, 128 mg.; cholesterol, 173.8 mg.; carbon dioxide, 57 per cent. A study of the calcium showed a slight increase in the output for a total of twenty-four hours. Studies of the sugar tolerance of the blood and urine revealed for the blood, one-half hour, 245.1 per cent; one hour, 300 per cent; two hours, 288.4 per cent; three hours, 214.3 per cent; for the urine, one-half hour, 0; one hour, 0.4 per cent; two hours, 0.7 per cent; three hours, 0.3 per cent.

The basal metabolic rate was minus 7 per cent. Studies of hormones in the urine revealed that prolan A was absent and that the ovarian hormone equaled 4 rat units. Roentgenograms revealed a slight atrophy of the posterior clinoid processes; atrophy of the cancellous portion of the bodies of the dorsal vertebrae, though the margins appeared quite dense, and a similar atrophic change of the lumbar vertebrae. The uterus was of normal size, and the adnexa was not felt.

Surgical Diagnosis.—Pilonidal cyst; excision was advised.

Course.—Roentgen therapy was administered, and there has been no material change in the patient's condition, except for a return of the menses.

Comment.—In this case the history and prognosis were characteristic of a basophilic adenoma of the pituitary gland. Development of the marked abdominal adiposity, with radiating striations, had not yet occurred; there was neither hypertension nor erythremia. The other signs are customary. It was of additional interest that the cholesterol was at the high limit of normal, and that the output of calcium was increased. These observations are comparable with those noted in one of Cushing's cases. The absence of prolan A and the accompanying low excretion of ovarian hormone were noteworthy.

CASE 3.—*Hypertrichosis of the face, hypomenorrhea, adiposity, vascular hypertension, headaches, pains through the body, purpuric ecchymoses and erosion and enlargement of the sella turcica.*

History.—A. A., single, aged 38, was referred to the Vanderbilt Clinic by Dr. I. H. Semken in October, 1932, complaining of increase in weight, though she had always been obese since a ventral suspension of the uterus with partial oophorectomy in 1919. She had had frontal and occipital headaches for four years. There had been spells of confusion with feelings of fainting for nineteen months. Black and blue spots had appeared readily on the body for about two years, and she had had pain through the body fat. Facial hair, though always present, had

Fig. 7 (case 3).—Patient with short square build and generalized obesity.

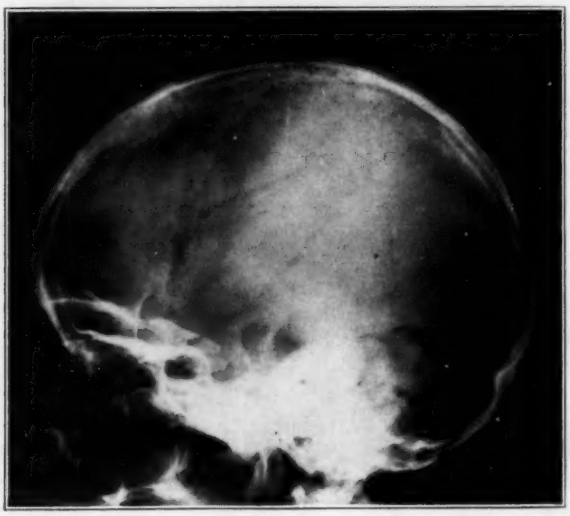

Fig. 8 (case 3).—Roentgenogram of the skull, showing the depressed sella turcica with thinning of the dorsum and posterior clinoids.

grown excessively in the past year. She also complained of somnolence. In 1931 she had received treatment for essential hypertension at the Cornell Clinic. The menses began at 14, with a cycle of from thirty to thirty-two days, and lasted only one day. Lately they had appeared at longer intervals and had been more scanty. For the past year the breasts had been painful for two weeks before the periods.

Examination.—The patient was 4 feet and 8 inches (142 cm.) tall; the torso-leg ratio was 47:70; the build was broad; the fingers were tapering; fat was generalized throughout the body, and there were a beard, a moustache and feminine pubic hair. White striae were present on the hips, abdomen and breasts. The fundi were normal; the fields were contracted on repeated examination, with no quadrantic defects.

The blood pressure was 170 systolic and 90 diastolic; the pulse rate was 80. Examinations of the blood revealed: hemoglobin, 86 per cent; red cells, 4,760,000; white cells, 7,000; polymorphonuclears, 73 per cent; lymphocytes, 27 per cent; urea, 13 mg.; sugar, 99.3 mg. per hundred cubic centimeters; tests for sugar tolerance showed 168, 157, 147 and 117 mg. in the blood with no glycosuria. The basal metabolic rate was plus 5 per cent.

A roentgenogram of the skull showed the sella turcica to be deformed. The floor was depressed and thinned out, with increased depth of the sella and thinning of the posterior clinoids and dorsum. The anterior clinoids were not abnormal. The findings suggested a pituitary adenoma (Dyke). The spine showed no decalcification or compression of the vertebrae. An encephalogram showed large ventricles, but otherwise nothing noteworthy.

Course.—Irradiation of the pituitary gland has been followed by definite improvement of the headaches.

Comment.—In this case there were definite signs of an intracranial lesion: headache, constriction of the visual fields and destruction of the sella turcica. On the other hand, there were certain characteristics indicative of a basophilic syndrome, i. e., masculine growth of hair, vascular hypertension, obesity, menstrual deficiency and purpuric ecchymoses. I believe that this patient has an adenoma of the basophilic type, though it may be a mixed adenoma of the pituitary gland. The previous diagnosis of essential hypertension is of special interest because of an important relationship to the basophilic syndrome.

CASE 4.—*Hypertrichosis with beard, vascular hypertension, hypomenorrhea, obesity, striae, headaches and polyuria.*

History.—J. B., married, aged 41, was referred to the Vanderbilt Clinic by Dr. Josephine Kenyon in March, 1932, because of obesity and the fact that her brother was receiving treatment for pituitary disease. She was easily fatigued and since the age of about 14 had had intratemporal headaches which were worse for a time. They were accentuated before the menses. Growth of hair on the face had begun at about 20, and she now shaved daily. For the past six months she had noticed edema of the lower extremities, swelling of the abdomen and constipation. At times, without apparent cause, polyuria and diarrhea appeared suddenly and she lost many pounds in several days. The menses began at 15, and were always delayed and scanty, often skipping from six weeks to two months. Lately they had occurred every twenty-eight days and had lasted three days.

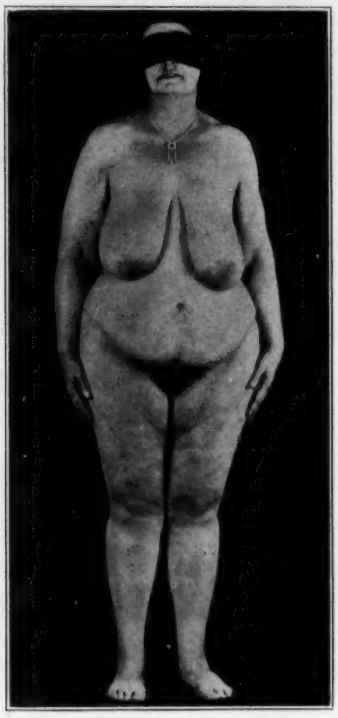

Fig. 9 (case 4).—Patient with girdle obesity.

Fig. 10 (case 4).—Roentgenogram of the skull, illustrating the bridged sella turcica.

Basal metabolic rates of minus 20 and minus 5 per cent had been reported, and the patient had received treatment with a thyroid preparation for two years.

Examination.—The torso-leg ratio was 49:85; the fingers were tapering; the skin was dry; there were hypertrichosis on the body, and a moustache and beard. There were an obesity girdle and much fat above the knees. Striae were marked on the abdomen, breasts, thighs and arms. The fields of vision were normal.

The blood pressure was 180 systolic and 90 diastolic; the pulse rate was 104. The blood contained: hemoglobin, 88 per cent; red cells, 4,800,000; sugar, 103 mg.; calcium, 9.4 mg. The Wassermann test was negative. The basal metabolic rate was minus 6 per cent.

A roentgenogram of the skull showed the sella turcica to be of average size, with a bridge between the anterior and posterior clinoids. The spine showed hypertrophic spondylitis and some arthritis.

Comment.—In this case also there are many of the symptoms of the basophilic syndrome, though the tendency to purpura, the characteristic adiposity and osteoporosis are lacking. Nevertheless, it is a case of pituitary disease in which the most marked changes suggest involvement of the basophil cells. There is no evidence to suggest the development of a neoplasm. However, headaches have been increasingly distressing and may indicate the beginning of a hypophyseal enlargement.

CASE 5.—*Hypertrichosis (masculine type), rapidly acquired obesity, vascular hypertension, amenorrhea, low sugar tolerance, frontal headaches and exhaustion.*

History.—P. S., single, aged 18, was admitted to the Vanderbilt Clinic in May, 1932, complaining of severe frontal and occipital headache, pains in the upper quadrant of the abdomen, fainting spells and exhaustion, increasing growth of facial hair for two years, and a rapid gain in weight at the age of 12 or 13. The menses began at 13 and were regular for two years; then they were spaced at longer intervals until at the time the patient was seen they occurred only at nine or twelve month intervals.

Examination.—The height was 65½ inches (166 cm.); the weight, 330 pounds (149.68 Kg.); the torso-leg ratio, 51:89. A large broad build and a broad jaw with spaced teeth were noted. There was slight kyphosis, and the fingers were tapering. Obesity was excessive throughout; the breasts were small. There was hypertrichosis on the body, also a nasal brow, moustache and masculine pubic hair. Gynecologic examination revealed a male type of pelvis; pigmentation of the labia was increased, and the clitoris was well developed. The blood pressure was 170 systolic and 130 diastolic.

The blood contained: hemoglobin, 83 per cent; red cells, 4,900,000; white cells, 12,000; polymorphonuclears, 76 per cent; urea, 12 mg. The Wassermann test was negative. The curve for blood sugar was 104, 214, 254, 230 and 187 mg. per hundred cubic centimeters, with urine sugar of 0.8 and 1 per cent. The basal metabolic rate was plus 10 per cent.

Roentgenograms of the skull showed an irregularity of the inner table; the sella was heavy and not enlarged, but of average size; the pineal gland was calcified. The skull appeared acromegalic. The spine showed no changes.

Comment.—In this case the sequence of events indicates a profound endocrinopathy developing progressively and beginning with the onset

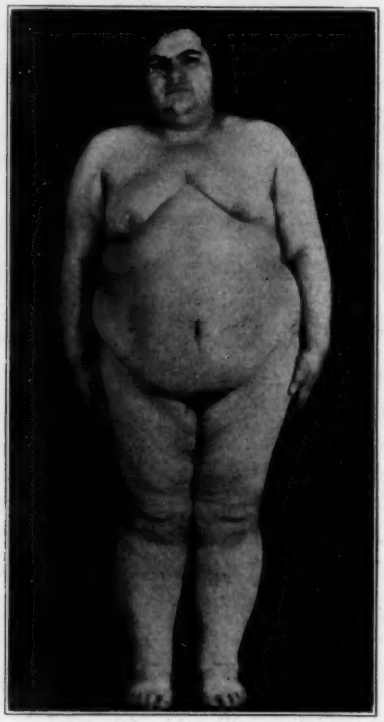

Fig. 11 (case 5).—Patient with moustache, beard, small breasts and excessive obesity.

Fig. 12.—Roentgenogram of the skull, showing the heavy sella turcica and general acromegalic type.

of puberty. The basophilic syndrome of the pituitary type is present, but it is also probable that there is an accompanying adenoma of the suprarenal cortex, a not infrequent concomitant, which may account in part for the virilism and explain the periodic attacks of acute pain in the upper part of the abdomen. The confusion between these two syndromes and the frequency with which a hyperpituitary and hypersuprarenal syndrome appear together make a separation of their symptomatology almost impossible.

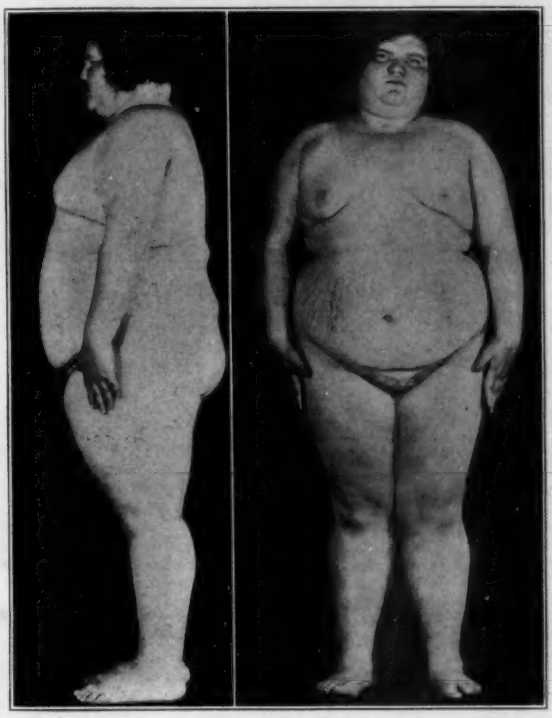

Fig. 13 (case 6).—Marked obesity and purplish lineae atrophicae are present.

CASE 6.—*Rapidly acquired plethoric obesity, purplish lineae atrophicae, vascular hypertension, erythremia, hypomenorrhea, headache and beginning osteoporosis (?) of skull.*

History.—H. G., single, aged 16, was admitted to the Vanderbilt Clinic in November, 1932, complaining of gradually developing obesity since 10 years of age and a present weight of 285 pounds (129.27 Kg.). She had fainted on the day before admission; she suffered from frontal headache (intertemporal). Tonsillectomy was performed at 14. The menses began at 12 and came every four or five months, lasting four days; the periods were preceded by frontal headaches and cramps.

Examination.—The build was large and broad, with generalized fat. Hair was present on the body but was not excessive; there was no facial hair. The hands

and face were strikingly plethoric and acrocyanotic. Purplish lineae atrophicae were present on the shoulders, chest and abdomen. The blood pressure was 140 systolic and 60 diastolic, and the pulse rate was 100.

The blood contained: hemoglobin, 88 per cent; red cells, 5,150,000; white cells, 10,000; polymorphonuclears, 53 per cent; lymphocytes, 47 per cent. The Wassermann test was negative. The basal metabolic rate was plus 9 per cent.

Roentgenograms of the skull showed a slight degree of porosity of the inner table of the parietal bones and no evidence of increased pressure. The sella turcica measured 11 mm. in the anteroposterior diameter. The dorsum was hazy, as were the posterior clinoids, suggesting a mild atrophy.

Comment.—In this case the grouping of symptoms and signs strongly suggests a predominance of the influence of the basophil cells of the anterior lobe. No evidence is present even to suggest a neoplasm, though

Fig. 14 (case 6).—Roentgenogram of the skull, showing an average-sized sella turcica and a hazy dorsum and clinoids suggesting a mild atrophy.

headaches, pituitary in type, indicate some activity within the sella turcica. I am not inclined to believe that this patient has a basophilic adenoma, but she should be watched.

CASE 7.—*Rapidly advancing adiposity, hypertrichosis, purplish lineae atrophicae, slight hypertension, intertemporal headaches, porosity of inner table of the skull, precocious sexual maturity.*

History.—S. L., a boy, aged 14, was admitted to the Vanderbilt Clinic in December, 1932, with the complaint of a general gain in weight since the removal of the tonsils at the age of 3, and a gain of 40 pounds (18.14 Kg.) in the past two months. When seen he weighed 222 pounds (100.69 Kg.). He complained of intertemporal headache and fondness of sweets. The father and mother both weighed over 200 pounds (90.72 Kg.).

Examination.—The patient was obese, especially about the abdomen and hips, with purplish lineae atrophicae on the thighs; the thyroid was full and boggy;

Fig. 15 (case 7).—Mature growth, hypertrichosis and adult sex organs are seen.

Fig. 16 (case 7).—Roentgenogram of the skull, showing porosity of the inner table of the skull with a small enclosed sella turcica.

there was hypertrichosis, with a nasal brow and marked growth of hair on the face, arms, chest, thighs and abdomen; the central incisors were broad and spaced. The genitalia were of adult form; the penis was well developed and implanted in an adequate scrotal region. The blood pressure was 140 systolic and 90 diastolic; the pulse rate was 70.

A roentgenogram of the skull showed a tendency toward porosity in the inner table and extending through the diploe. The sella turcica was small and shallow, and the dorsum inclined somewhat forward; its anteroposterior diameter was 12 mm., but its capacity was below the average for the size of the skull. Roentgenograms of the spine disclosed no abnormalities. The basal metabolic rate was minus 2 per cent. The blood picture was normal.

Comment.—In this case the signs are obviously of pituitary origin; together they indicate a predominant activity of the basophil cells because of the increased blood pressure, hypertrichosis and purplish striae in a boy at the age of puberty. Whether this condition will progress and whether further signs of an adenoma will develop, only further observation will reveal. It is not at all improbable.

Summarizing the seven cases, it appears that all present more than half of the characteristic features of the basophilic syndrome. All showed adiposity; one was of the abdominal type originally described by Cushing. Amenorrhea was present in two patients (cases 2 and 5), and three (cases 3, 4 and 6) showed marked diminution and infrequency of the menses. Hypertrichosis was present in all the patients except one (case 6). Purplish lineae atrophicae and plethoric or purpuric spots were present in five (cases 1, 2, 3, 6 and 7). Vascular hypertension was present in all except one (case 2). Polycythemia was present in two patients (cases 1 and 6). Osteoporosis was present in three (cases 1, 3 and 6 [?]). Headaches, fatigability and pains were present in all the patients. Disturbance of sugar metabolism (low tolerance) was found in two patients (cases 2 and 5). Three (cases 1, 2 and 3) showed signs indicative of intracranial pressure, such as a disturbance of visual fields, exophthalmos and polyuria. The age of these patients, except in two cases, averaged about 18 (from 14 to 24 years). All were of short stature except one (case 5), and the trunk-leg ratios, when noted, were striking in that the torso far exceeded the proportionate relationship to the legs.

It seems obvious, therefore, that all of these patients present a certain number of features which permit one to classify them as exhibiting a basophilic syndrome of the pituitary gland. In five cases (cases 1, 2, 3, 4 and 5) there was sufficient evidence to suggest strongly the presence of a basophilic adenoma. Only future observations will show whether this assumption is justified.

COMMENT AND CONCLUSIONS

The syndromes of pituitary basophilism are numerous and at the present time can only be tentatively outlined.

1. The Cushing syndrome, with a rapidly developing basophil adenoma, terminating, in most reported cases, in death within five years. The clinical picture of this syndrome is a characteristic one with well marked physical signs (cases 1 and 2).

2. A mixed syndrome of intrasellar pituitary disease, with evidence of a neoplasm. These patients present features of basophilism, though less marked and less numerous than those in group 1. They may also present signs of acromegaly and an evidence of acidophil cellular overactivity (case 3); this combination has also been reported by Cushing.[3] One sometimes sees evidences of both basophil and acidophil cellular influence in patients who do not show any demonstrable evidence of adenoma.

3. A syndrome in which the disturbances appear to point to involvement of the suprarenal cortex (as measured by old standards), but in which with more complete understanding of the basophilic syndrome one may consider a possible involvement of both the suprarenal and the pituitary glands. Case 5 demonstrates this group well; with difficulty I have saved the patient from an exploratory operation on the suprarenal glands.

4. A prepubertal or pubertal basophilic syndrome. This is a disturbance of developmental growth in which there is precocious sexual development, associated with evidences of pituitary basophilism. Rarely do these cases pass into group 1; more commonly compensation takes place and they reach a static phase compatible with health (similar to the process occurring in patients with pituitary overgrowth—acromegaly or gigantism—who become stabilized and symptomless, an acidophilic syndrome). Cases 6 and 7 of this series demonstrate this group well.

5. Postmenopausal basophilic syndrome. This includes the group of women whose ovarian activity has ceased, with the development in pituitary gland of so-called "castration" cells. These cells, largely basophilic, may be the cause of the production of such signs, which one so frequently sees, as pituitary headache, hypertrichosis with a beard, hypertension, obesity, hyperglycemia and often associated thyroid and suprarenal signs. No doubt this group is the precursor of the "bearded old woman" type.

It has been my desire to confirm the characteristic syndrome of pituitary basophilism described by Cushing and to demonstrate that all basophilic syndromes are not necessarily progressive and fatal; likewise,

3. Cushing,[1] p. 119, note 15.

that pituitary adenomas presenting many features of this syndrome exist, and are either pure pituitary basophilism or combined with acidophilism and disease of the suprarenal glands; also, that "transitory or mild degrees of pituitary basophilism" (quoting Cushing [4]) do exist, not only in adolescents but in premenopausal and postmenopausal states.

One need not inquire what produces an adenoma of the basophil cells, for the solution of the problem of tumors is still far removed. But what does produce a predominant activity of these cells? The close relationship of the pituitary gland to the gonads is, no doubt, an important factor, because this syndrome occurs more frequently in women. It has been affirmed that the basophil cells produce the gonad-stimulating hormone of the pituitary gland (anterior lobe). This is an assumption which is not yet well founded and which will probably be altered on both experimental and practical grounds. Both case 1 and case 2 of my series showed an absence of prolan in the urine, as did one of Cushing's cases. These patients present every evidence of underactivity of the sex function rather than a stimulatory effect. One will have to fall back on a theoretical explanation which presumes that a proper balance between the endocrine organs has been disturbed, resulting in a pluriglandular disorder, the outstanding feature of which is the basophilic syndrome.

DISCUSSION

DR. HARVEY CUSHING, Boston: I have listened to Dr. Pardee's contribution with great interest and at the same time with some apprehension. I judge the first two of the reported cases to be unmistakable examples of what I have ventured to call "pituitary basophilism." But I feel far less sure of the others. What makes me apprehensive is Dr. Pardee's attempt to divide this syndrome into a number of subvarieties when, after all, little is known about the complete expression of the disease. Certainly the more that is learned about it, the less inclination there will be to divide it into clinical subtypes.

What is chiefly needed for the progress of knowledge is a greater number of pathologically verified cases and less clinical speculation regarding the etiology of suggestively similar and borderline states. I am sure that Dr. Pardee will agree with me.

All pituitary disorders are, of course, symptomatically polyglandular, and the variable degree in which they secondarily derange the parathyroid, suprarenal, thyroid glands or pancreatic islets will serve to modify the resultant clinical picture. In the case of acromegaly, the clinical manifestations of which are no less polyglandular than those of pituitary basophilism, one does not gain much by classifying all its clinically expressed subvarieties. When pituitary basophilism comes to be equally well understood, it will be looked on much as acromegaly is, namely as an entity characterized by a variably expressed symptom complex.

So far, it has been possible to gather from the literature fifteen cases showing a fully developed syndrome and associated with a proved adenoma of the pituitary gland, which in eleven instances was definitely basophilic. By a basophilic adenoma

4. Cushing,[1] p. 159.

I mean what the term "adenoma" implies and not merely, as some critics have assumed, the tiny accumulation of basophilic elements so often seen in the pars distalis.

It is known with reasonable certainty that the acidophilic elements produce the hormone of growth and that the adenoma accompanying gigantism and acromegaly is invariably composed of these elements. What one needs to learn is whether the gonadotropic hormone, when separately isolated from the growth hormone, can reproduce the clinical syndrome under discussion if given to animals over long periods. If this is possible, it will be the first definite proof that the basophilic elements are actually the source of the gonadotropic hormone. What is more, it will provide a means of studying this peculiar derangement in all its polyglandular manifestations.

I hope that Dr. Pardee will not take amiss my suggestion to proceed slowly with the subvarieties of this disorder until more definite knowledge is obtained regarding it, lest the issue be confused rather than clarified. I speak with feeling on this subject, as I have made one or two regrettable mistakes myself in predicting a basophilic adenoma in cases in which none was found.

Dr. Irving Pardee (New York): In setting the term of life for these patients at five years, I was quoting Dr. Cushing's contribution on the subject in which he reported ten fatal cases with an average length of life of five years. Of course, none of the patients I have cited has died. In all the development of pituitary basophilic signs is still going on. The first case is unquestionably well developed; the second shows characteristics which closely approximate those of the first, although there is no high blood pressure. In the other patients there is evidence of pituitary disease with an associated basophilic syndrome. The last two cases occurred shortly after puberty, and later a complete picture of pituitary basophilism may develop.

I wish to bring out the endocrine signs these patients present in the hope that by recognition of those signs these patients can be helped before they are ready for a postmortem examination. I believe that by noting such signs as menstrual disturbances, hypertension, hypertrichosis, etc., one will soon find that there is a large group of patients presenting the basophilic syndrome, in whom the outcome will not be fatal but who indicate a predominant activity of the basophil cells without the presence of an adenoma.

THE THIRD VENTRICLE

CONFORMATION OF THE FLOOR AND ITS RELATION TO THE MENINGES

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Neurologists have recently focused considerable attention on the third ventricle, but their interest has been confined largely to a search for important autonomic nuclei believed to be located within its walls. Less attention has been paid to the structure of the ventricle itself and to its relation to the meninges and their cisternae. It has been largely overlooked that the floor of the third ventricle is so thin as to constitute a vulnerable point at which the slightest lesion may penetrate the ventricle and permit the cerebrospinal fluid to flow freely into the surrounding cisternae. Dandy [1] long ago made practical use of this fact in the treatment of internal hydrocephalus by puncturing the floor of the ventricle so that the fluid might drain into the cisterna interpeduncularis and the cisterna chiasmatis. Investigation of the factors involved in the production of diabetes insipidus led us to study in more detail the conformation of the floor of the third ventricle and its relation to the meninges. We have come on several interesting structural features and have been able to show not only that the floor is much thinner than is commonly supposed, but that, at least in one place, it pouches out over a fairly large area on the surface of the brain.

The observation which led us to make the present investigation can be stated simply. It was found that permanent diabetes insipidus could be produced in rats by a stab wound with a small scalpel through the sphenoid bone and into the brain near the anterior margin of the pituitary gland (Richter [2]). No definite conclusion could be drawn from these experiments, however, owing to the fact that the wounds involved not a single structure but several different ones. The stalk of the pituitary gland was either partially or totally severed; nuclei in the walls of the third ventricle were injured or their pathways interrupted, or nervous and circulatory connections between the brain and the pituitary

From the Psychobiological Laboratory, Phipps Psychiatric Clinic, Johns Hopkins Hospital.

1. Dandy, W. E.: Bull. Johns Hopkins Hosp. **33**:189, 1922.
2. Richter, C. P.: Brain **53**:76, 1930.

gland were cut. For this reason a second series of experiments was undertaken with the object of determining the importance of each of these different factors.

It was known from earlier extirpation experiments (Richter and Wislocki [3]) that in some instances the pituitary gland can be completely removed without disturbing the water metabolism of the body, while in other cases diabetes insipidus follows. As the sphenoidal approach to the pituitary gland and the suction method of removal eliminated the possibility of any damage to the deeper parts of the brain, the conclusion was drawn that the structure responsible for the production of the polyuria must be located near the surface of the brain, in such a position that it might be injured during the operation. It seemed possible, moreover, that the cerebrospinal fluid might be involved, since it often flowed more freely after the gland was removed.

In order to illuminate this aspect of the problem we endeavored by injection methods to determine the exact relation between the third ventricle and the regions with which we were concerned. It was found that india ink injected into the third ventricle of a freshly removed normal brain of a rat appeared at once at the surface of the brain just posterior to the pituitary stalk, in a saclike, leaf-shaped structure pointing away from the gland and extending between the mamillary bodies. An investigation of this interesting structure was undertaken, on its own merit as well as in connection with the problem of diabetes insipidus, and the present paper contains a description of it as it is found in several animals—the rat, guinea-pig, rabbit, porcupine, cat, dog and monkey and in man. At the same time, the conformation of other parts of the floor of the third ventricle was studied, with particular reference to the relation of these parts to the surface of the brain and to the encasing meninges.

METHODS

In the present experiments it was important to remove the brain without injuring the meninges or the walls of the third ventricle and to inject india ink or methylthionine chloride, U. S. P. (methylene blue) in such a way that it would fill the most remote recesses of the ventricle.

We found that the most effective method of removing a rat's brain without tearing the meninges was to start from the ventral surface at the sphenoid bone and to work around both sides until the entire brain was uncovered. In this way it was possible to remove the brain with the dura intact over the pituitary gland and the immediately surrounding structures.

A drop of india ink or of methylene blue was injected under very slight pressure through a glass cannula inserted into the brain at the lamina terminalis in the midline, just anterior to the optic chiasm. The point of insertion of the cannula may be seen in figure 1 *A*, a photograph of a midline sagittal section of a rat's brain. The entrance of the material into the ventricle could be seen at once

3. Richter, C. P., and Wislocki, G. B.: Am. J. Physiol. **95**:481, 1930.

through the thin walls of the pituitary stalk in the floor of the ventricle. After the ink had been injected, the dura over the pituitary gland was removed, and the gland, freed from its attachments on both sides, was folded back on the stalk. In this way the tuber cinereum and the surface of the mamillary bodies were exposed to view, while the arachnoid and pia mater, with the enclosed cisternae, were still intact.

RESULTS

Rat.—The saclike structure already mentioned can be seen in the photographs in figures 1 *B* and *C* and 2 *A*, in which it is designated as area 1. In figure 1 *B*, showing a ventral view of a rat's brain with the hypophysis removed, it can be seen that this leaf-shaped or arrow-shaped area lies posterior to the infundibulum between the tuber cinereum and the mamillary bodies. It is about 0.8 mm. long, 0.8 mm. wide and 0.1 mm. thick. It often comes to a fine point and extends as a threadlike structure for a considerable distance along the surface of the brain between the mamillary bodies. In the section shown in figure 1 *C*, it can be seen that it is in close proximity to the surface of the brain. Further orientation may be obtained from the sagittal section in figure 1 *A*, in which the location of the leaf area just ventral to the mamillary bodies is demonstrated.

It is important to note that this area is rarely seen in a brain into which dye has not been injected, although it may be slightly visible when there has been bleeding into the ventricle. It appears in all normal rat brains, however, when minute amounts of india ink or methylthionine chloride are injected. It is not possible to produce a rupture at this point in normal animals even when the injection is made under considerable pressure, owing to the fact that the walls seem to give way more easily elsewhere.

Two neighboring regions with thin walls were located by means of the injection method: area 2, posterior to the infundibulum at the point of junction of the infundibulum and the brain, and area 3, anterior to the infundibulum on the floor of the ventricle. Area 2 can be seen in figure 2 *A*, a photograph of the ventral surface of the brain with the pituitary gland folded on its stalk, exposing the tuber cinereum. This area appears only when the material is injected under great pressure and consists merely of a rupture of the wall of the third ventricle at the point where the ventricle extends into the infundibulum. Such a rupture can easily be produced when the gland is lifted away from the brain and often means a severance of connection between the posterior lobe and the brain. When the ink or dye is injected under even higher pressure, a rupture is often produced in the brain tissue between areas 1 and 2, as seen in the threadlike structure shown in figure 2 *A*.

Area 3 can be seen in figure 1 *B*. It is usually slit-shaped; it lies anterior to the infundibulum and extends from the infundibulum forward to the end of the tuber cinereum. As the stalk of the hypophysis is maintained as a distinct structure for a short distance anterior to its junction with the brain, this slit-shaped area indicates a separation of the stalk into halves. Ink injected into the ventricle in minute amounts is always visible at this place, but when injected under high pressure it becomes very conspicuous, as may be seen in figure 1 *B*. Rupture occurs less easily here, however, than at area 2.

Since these areas were observed consistently in the rat's brain, a search was made for them in several other animals.

Guinea-Pig.— In figure 2 *B* it is apparent that the leaf area (area 1) and area 2 are both present in the guinea-pig. Area 1 is located almost at the base of the

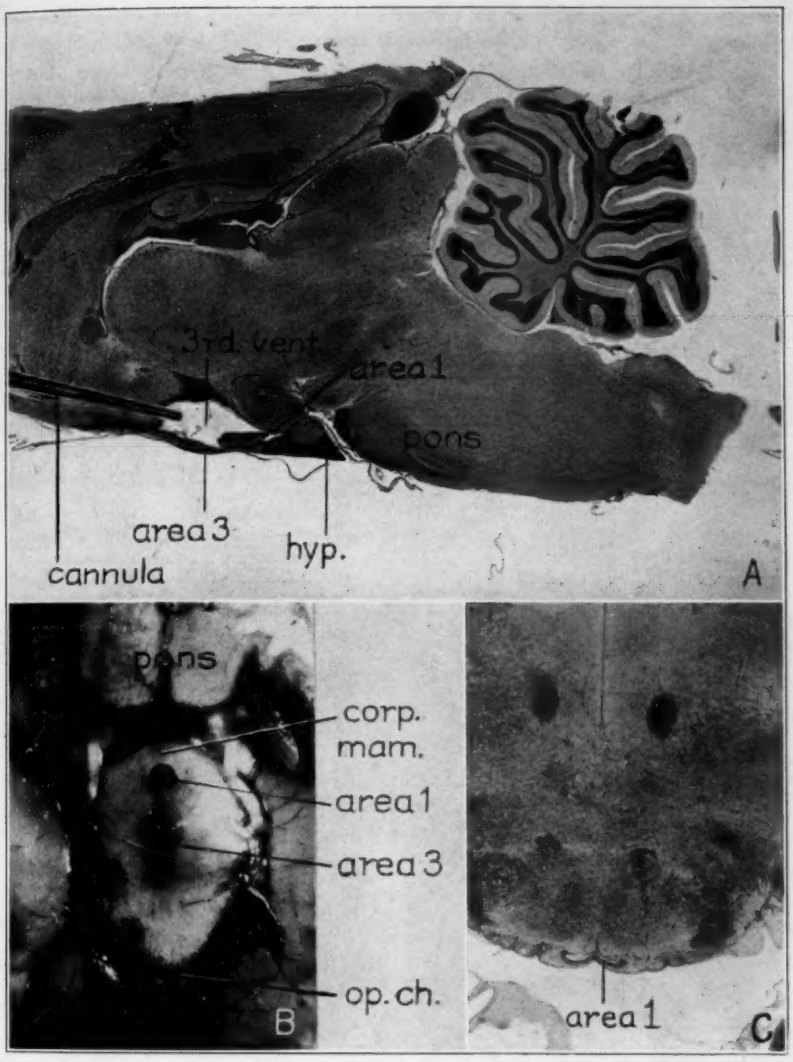

Fig. 1.—*A*, a midline sagittal section of a rat's brain, showing the relation of the hypophysis to the third ventricle. *B*, a ventral view of a rat's brain into which ink has been injected and from which the hypophysis has been removed. The photograph shows area 1 located posterior to the infundibulum, near the dividing line between the tuber cinereum and the mamillary bodies, and area 3, slit-shaped, anterior to the infundibulum, extending along the most ventral part of the floor of the ventricle. *C*, a transverse section of a rat's brain through area 1.

stalk and is more circular than in the rat; area 2 appears in the infundibulum. The close proximity of these two regions is demonstrated well in sagittal sections. Area 3 was not seen in any of the guinea-pig brains studied.

Rabbit.—In the rabbit area 1 was found to be larger than in the rat, but in approximately the same relative location. Considerable difficulty was encountered in obtaining intact specimens. The one depicted in figure 2 *C* shows area 1, but

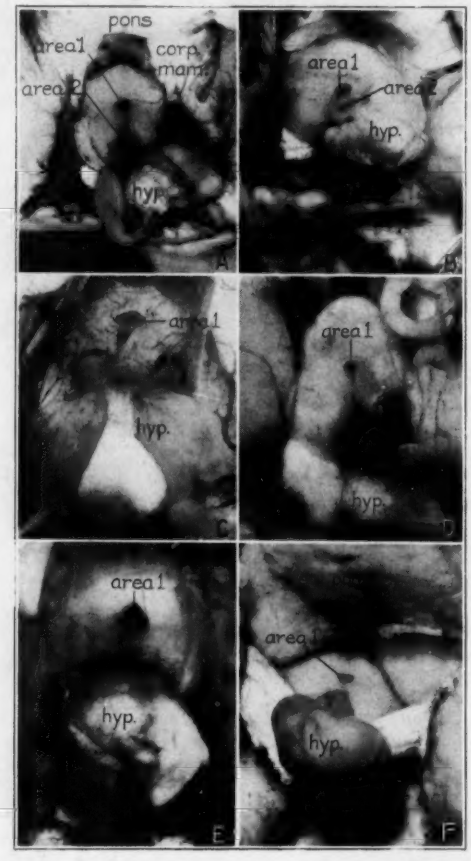

Fig. 2.—Ventral views of the brains of various animals, with the hypophysis lifted away from the brain and folded on its stalk, exposing the leaf area in the region of the tuber cinereum. *A*, rat's brain, showing areas 1 and 2. Area 2 is located at the junction of the stalk and the brain. *B*, guinea-pig's brain, showing areas 1 and 2. *C*, rabbit's brain, showing area 1. *D*, porcupine's brain, showing area 1. The ink has diffused out through area 2. *E*, dog's brain, showing only area 1. *F*, monkey's brain, showing only area 1.

with a wedge-shaped addition on the anterior part extending to the infundibulum. This wedge-shaped addition, we believe, was the result of a rupture of the wall produced by excessive pressure of the injection fluid. Areas 2 and 3 were not seen in these animals.

Porcupine.—In the porcupine, area 1 appears as a very small triangle at a considerable distance from the infundibulum (fig. 2*D*). Serial sagittal sections showed that in this animal, in contrast to the others studied, the floor of the ventricle is separated from the surface of the brain by a thick wall all along the tuber cinereum. Rapid inspection failed to reveal the out-pouching of the ventricle found

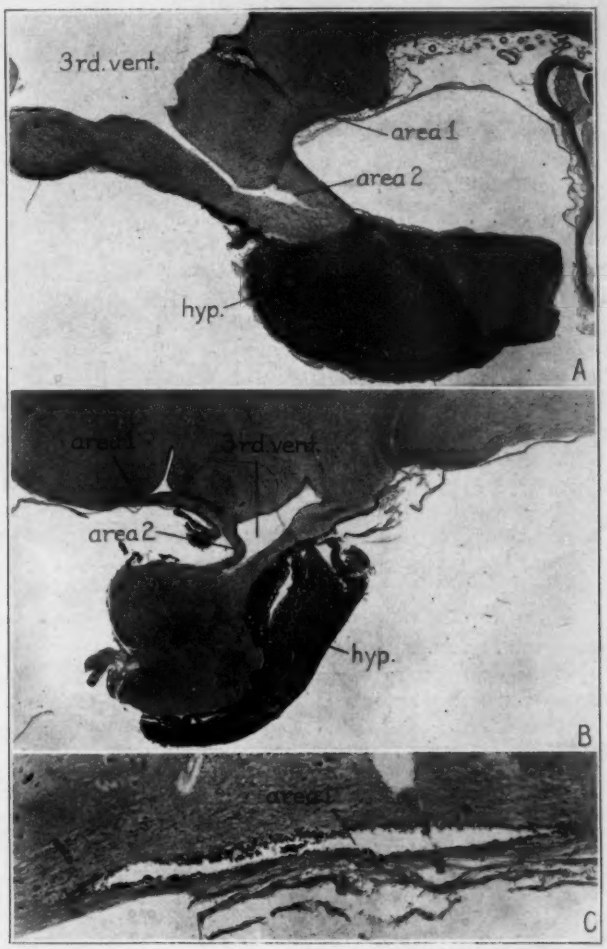

Fig. 3.—*A*, a sagittal section of a porcupine's brain, showing the channel connecting the third ventricle and area 1. *B*, a sagittal section of a dog's brain, showing part of area 1 and areas 2 and 3. *C*, a transverse section through area 1 of a dog's brain, showing the lining of ependymal cells and the close proximity of this area to the pial membrane.

so readily in the other animals, but a more detailed study showed the structure in a few sections, one of which is presented in figure 3*A*. It can be seen that there is a small, thin channel from the ventricle through the wall to the surface of the

brain, just where it would be expected from the location of the leaf area in figure 2 *D*. Under a higher magnification this channel was found to be lined with the usual ependymal cells.

The isolated nature of area 1 in the porcupine suggests strongly that it is more than a simple extension of the ventricle—a special structure perhaps, as will be mentioned later.

In the preparation photographed in figure 2 *D*, the ink had diffused out through area 2 at the base of the stalk. We had only a few porcupines at our disposal and did not succeed in removing the brain of any of them with this area intact. Area 3 was not observed in these animals.

Dog.—In the dog the leaf area is striking. It may be seen in figure 2 *E* emerging on the boundary line between the tuber cinereum and the mamillary bodies, just as it does in the rat, and its shape is much the same. A sagittal view of area 1 and area 2 can be seen in figure 3 *B*.

The relation of the leaf area to the meninges is clearly demonstrated in the section made transversely in this region in the dog (fig. 3 *C*). The sac, lined with the usual ependymal cells, is in almost immediate contact with the pia mater.

It proved extraordinarily difficult to obtain good injections in this area in the dog, because it is only with the greatest care and detailed knowledge of the anatomy of this region that the brain can be dissected with area 2 at the base of the pituitary gland intact. The slightest pull on the gland produces an opening not only into the subarachnoid space but in the arachnoid as well, and the injected fluid flows through this outlet rather than into the leaf area. Histologic studies have shown that a rupture of this area produced by pulling usually means the severance of a large part of the connections to the posterior lobe. Our experience has led us to conclude that it is almost impossible to lift the gland in operations near the hypophysis in the dog without severing this part of the stalk. This fact may be significant in a consideration of experiments in which various manipulations have been made in this region with the claim that the pituitary gland was entirely uninjured.

Cat.—Area 1 can be demonstrated in the cat, and the general relations are much the same as in the dog. However, it is even more difficult than in the dog to remove the brain without rupturing area 2, possibly because in the cat the pituitary duct is still patent and the infundibular canal extends into the gland for a much greater distance.

Monkey.—Area 1 in the monkey can be seen in figure 2 *F*. It has much the same shape and position as in the rat, the cat, and the dog. In the monkey, as in the cat and the dog, it is extremely difficult to remove the brain without injury at the base of the pituitary stalk. A leak of ink at this point, either into the subarachnoid space or out over the brain, completely obliterates the leaf area over the mamillary bodies.

Man.—In man, the same structures seem to be present, but they are rather markedly different in shape as well as in location. For purposes of orientation a ventral view of a human brain (from an infant 1 day old) is presented in figure 4 *A*. It can be seen that the mamillary bodies stand out separately and well away from the surface of the brain. In this respect they differ strikingly from the flat mamillary bodies of the rat, rabbit and other animals seen in figure 2. Furthermore, the surface of the tuber cinereum between the mamillary bodies and the infundibulum is not flat and smooth as it is in these animals but shows a distinct eminence extending from the anterior margin of the mamillary bodies to the infundibulum, which it joins almost imperceptibly. In further contrast to the brain

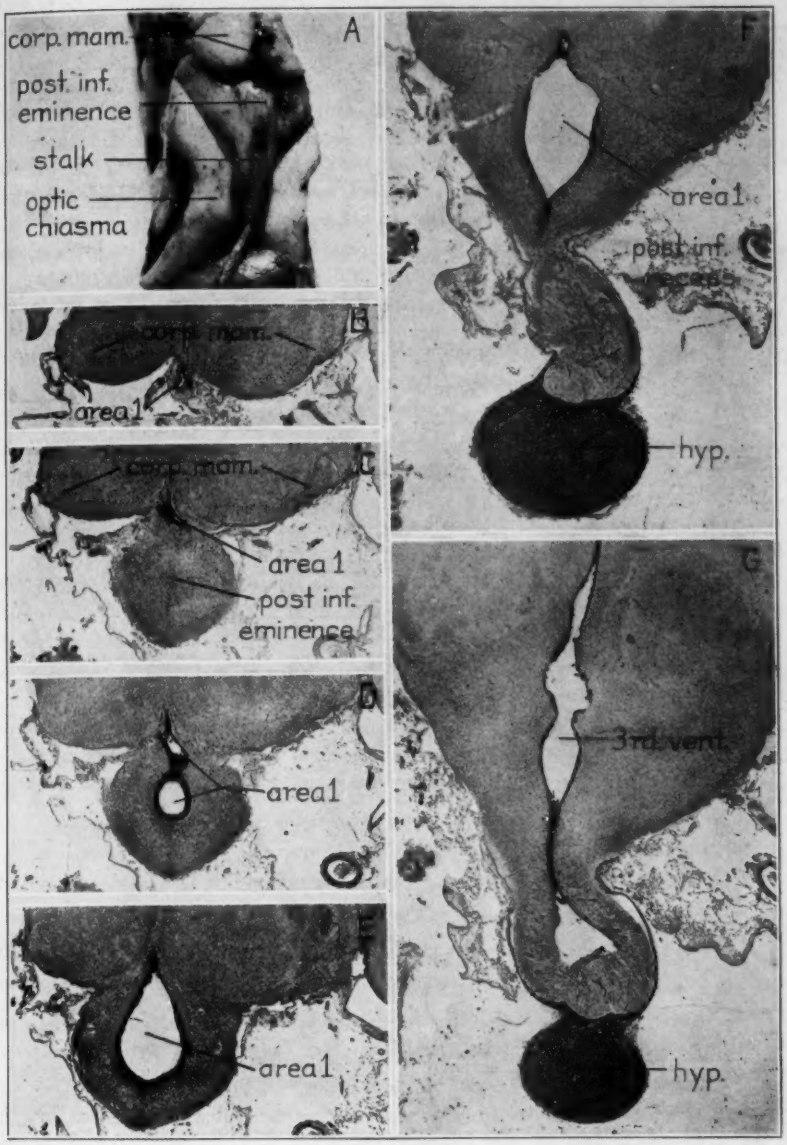

Fig. 4.—*A*, a ventral view of the brain of a new-born infant. Ink injected into the ventricle has leaked out between the mamillary bodies. *B*, a transverse section through the mamillary bodies of the brain of a new-born infant. *C*, a section through the posterior end of the postinfundibular eminence. *D*, a section made slightly farther anteriorly. *E*, a section made still farther anteriorly. *F*, a section made farther anteriorly near the insertion of the stalk. *G*, a section showing the third ventricle, stalk and pituitary gland of a human fetus.

of the rat and of the guinea-pig, the pituitary stalk of the human brain extends anteriorly rather than posteriorly, thus leaving the surface of the tuber cinereum and the mamillary bodies uncovered.

On the basis of these anatomic details the results of our observations can be discussed. Ink or dye injected into the third ventricle through the lamina terminalis causes a darkening of the tissue on the eminence posterior to the infundibulum, but there is no well circumscribed leaf area of the type seen in other animals. Further posterior in the eminence the ink comes to the surface in a small tube-shaped structure, where it continues for a short distance between the mamillary bodies. When the pressure on the injected material is increased very much this tubular structure is apt to rupture and thus permit the fluid to flow out freely. Thus it is seen that, although there seems to be some comparable structure in this region in the human brain, it is not circumscribed and does not come so near to the surface as it does in the other animals.

Histologic studies of this region of the brain disclosed the presence of a well defined saclike structure, which may be seen in figure 4. The first section (*B*), made through the anterior part of the mamillary bodies, shows a small tubular structure between the mamillary bodies, which in other sections can be seen to end blindly a short distance farther posteriorly. The next section (*C*), made farther anteriorly, shows the tubelike structure, now covered by the aforementioned eminence. In the next section, made still farther anteriorly, it can be seen how the tubular structure passes ventrally into the eminence. In the next section (*D*) the structure entirely fills the eminence. Section *E*, made a greater distance anteriorly, beyond the point where the stalk joins the brain, shows that the recess still stands out separately. In the last and most anterior section (*F*) the tubular structure has lost its identity and is a part of the main ventricle.

It has thus been found that in man the third ventricle has a long thin recess which passes into the postinfundibular eminence and extends to the mamillary bodies and for a short distance between them in the shape of a thin tubular structure.

COMMENT

His,[4] so far as we know, made the first reference to the diverticulum of the third ventricle which we have described. He stated merely that he had observed a saclike structure anterior to the mamillary bodies in human beings in early fetal life and that he believed it to be a homolog of the saccus vasculosus found in lower animals.

Later, this structure was described by Retzius[5] from a somewhat different aspect. He was interested in the external conformation of the brain and in extensive studies of animal fetuses observed a swelling in the surface of the tuber cinereum which extended bilaterally just anterior to the mamillary bodies and posteriorly for a short distance between them. He referred to the structure as a clover leaf, calling the two lateral parts *alae laterales* and the mamillary extension the *processus intermamillaris*. He found it in dogs, cats, reindeer, rabbits, guinea-pigs

4. His, W.: Arch. f. Anat. u. Entwcklngsgesch., 1892, p. 346.
5. Retzius, G.: Biol. Untersuch. **7**:1, 1895.

and human beings in early fetal life and in adult human beings and rabbits. In the brains of the other adult animals, according to the report, it was not present.

Retzius knew that this eminence is hollow and contains a recess from the third ventricle, but his knowledge of the shape and nature of this diverticulum was based, so far as we know, on what he could observe by external inspection and casual study of histologic sections. He knew, moreover, that in some animals the walls of the diverticulum are extremely thin, as we have shown. That he was able to learn so much about this structure is astonishing because, as has been pointed out, the leaf area is visible in brains into which no dye has been injected only when the cerebrospinal fluid is discolored by traces of blood. By injection methods he could have demonstrated the area in the brains of all mammals, adult as well as fetal.

The leaflike structure under consideration has also been described by Perna,[6] Staderini,[7] Sterzi [8] and Tilney.[9] In his extensive study of the conformation of the floor of the third ventricle, Tilney described an eminence with an underlying recess which he called the "postinfundibular eminence and recess." There can be little doubt that this structure is the same as that which we have described as the leaf area. Tilney's recess is seen most clearly in a published photograph of a sagittal section through the brain of a fowl (Gallus gallus).

The origin and function of this diverticulum are still unknown. His, as we have stated, thought that it was related to the saccus vasculosus seen in fishes, frogs and similar animals. Retzius also was of this opinion, and although he thought that more details were necessary before any definite conclusion could be drawn, he referred to the structure as the "saccular eminence," a term which has been adopted by several later writers.

Opposed to the hypothesis of a relationship between this sac and the saccus vasculosus are the views of Perna, Staderini and Sterzi, and most recently that of Tilney. Tilney supported the name "postinfundibular eminence and recess" in place of "saccular eminence and recess" on the basis of experiments which seem to demonstrate conclusively that the structure is entirely independent of the saccus vasculosus. He showed sections in which this structure and the saccus vasculosus were both present, but entirely independent and separated by a considerable distance. Our observations, particularly on the porcupine, are also definitely in favor of this view. In the porcupine the leaf area is located

6. Perna, G.: Arch. ital. di anat. e di embriol. **8**:597, 1909.
7. Staderini, R.: Arch. ital. di anat. e di embriol. **8**:116, 1909.
8. Sterzi, G.: Arch. ital. di anat. e di embriol. **3**:212, 1904.
9. Tilney, F.: J. Comp. Neurol. **25**:213, 1915.

at a considerable distance from the infundibulum and stands out independently but is still joined to the ventricle through this duct. It is not possible to make any conjecture regarding the probable function of this structure, chiefly because of the lack of detailed histologic knowledge. In all of our sections it appears to be lined largely with ependymal cells of the type seen in the other parts of the ventricle. It may be, however, that a detailed study will reveal other types of cells, as well as nervous or circulatory constituents. For the present the structure seems to have only a mechanical significance, as a thin part of the ventricle wall which, when injured, permits the escape of cerebrospinal fluid from the ventricle to the surrounding arachnoid spaces. One thing is clear, however—our pouchlike configuration is of an entirely different nature from the opening believed by Bichât [10] and Mierzejewsky [11] to exist in the walls of the fourth ventricle.

In conclusion, attention may be called once more to the fact that in all three of the areas described the cerebrospinal fluid of the third ventricle comes into almost immediate contact with the pia mater, the subarachnoid spaces and the cisternae. Any injury, however slight, to the surface of the brain at these places permits the fluid to flow freely to the outside. It is particularly with these facts that we have been concerned in our study of the experimental production of diabetes insipidus which will be reported in a later paper.

SUMMARY

The conformation of the floor of the third ventricle was studied by means of injections of india ink or methylthionine chloride made after the brain was removed.

It was found that the floor of the ventricle of the rat's brain comes into almost immediate contact with the pia mater in three places: Area 1 is a leaf-shaped, saclike structure in the postinfundibular region, between the tuber cinereum and the mamillary bodies. This area is present in all animals. Area 2 is an irregularly shaped area at the postinfundibular junction of the stalk and the brain. This area, present only when the ink is injected under excessive pressure or when the stalk is subjected to tension during dissection, is produced by a rupture of the canal that is traced up into the stalk. Area 3 is a slit-shaped area in the middle of the stalk between the infundibulum and the optic chiasm. Small amounts of ink can usually be seen through it, and it stands out strikingly when the injection is made under considerable pressure.

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10. Bichât, M. F.: Traité des membranes en général et de diverses membranes en particulier, Paris, Richard, Caille & Ravier, 1799-1800,

11. Mierzejewsky, J.: Centralbl. f. d. med. Wissensch. **10**:6, 1872.

Area 1 was found to be present in the rat, guinea-pig, rabbit, porcupine, cat, dog and monkey and in man.

Area 1, in the postinfundibular region, is of particular interest because it appears to be a saclike diverticulum from the ventricle, which in some animals, the porcupine particularly, is connected with the ventricle by only a very long thin channel. It is lined with ependymal cells and comes into immediate contact with the pia over a fairly large area. In the rat a threadlike extension from this part of the leaf continues for some distance between the mamillary bodies, as is also the case in man.

It was pointed out that this structure must be the same as that described previously by His, Retzius, Tilney and others on the basis of a study of the external conformation of the ventricles and referred to as the saccular recess or the postinfundibular recess.

It was stated further that, particularly on the basis of the work of Tilney, it has been established that this structure is not related to the saccus vasculosus of fishes and other animals. At this time we have no knowledge either of its origin or of its function.

EFFECT OF DIRECT STIMULATION OF BRAIN AND SPINAL CORD ON REFLEX TIME

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The results of experimentation already reported [1] attest the theory that stimulation of the higher neural centers produces an inhibitory effect on the lower neural centers and that inhibition of the higher neural centers produces a stimulatory effect on the lower neural centers. In the past our application of the action current technic to the study of reflex time has omitted controlled direct stimulation of the nerve tissues. In the present study we shall consider the effect of direct stimulation of the brain and the spinal cord on reflex response latencies.

In the stimulation of any tissue, tissues circumjacent to the point of application are distantly affected by the stimulus. In this study we have applied a technic for measuring the degree of radiation of electric stimuli in nerve tissues.

APPARATUS AND PROCEDURE

For clearness of presentation this experimentation may be divided into two major parts: experiments on dogs and experiments on rats. It is further helpful to classify the work according to the portion of the central nervous system that it involves. Thus there are series of experiments on the brain, the spinal cord and the peripheral nerves (sciatic).

Seven dogs and fourteen rats were surgically prepared under deep ether anesthesia. With the exception of one animal, only the particular area selected for study was exposed. In all animals the pia-arachnoid was left intact. In the experiments on the brain, the anesthesia had been discontinued for ten minutes in dogs and for ninety minutes in rats before the application of the stimuli. In the experiments on the spinal cord, the animals were kept under light ether anesthesia during the application of the stimuli. Three types of stimuli were used: pressure, acid and electricity. Pressure was applied by permitting a light silver plate to rest on the brain or spinal cord. A piece of gauze of the desired size was saturated with acid (1 per cent sulphuric acid) and placed over the area to be stimu-

From the Psychopathic Hospital, Ann Arbor, Mich., and the Psychopathic Hospital, Iowa City.

1. Travis, L. E., and Dorsey, J. M.: Effect of Alcohol on Patellar Tendon Reflex Time, Arch. Neurol. & Psychiat. **21**:613 (March) 1929; Patellar Tendon Reflex Time in Psychiatric and in Neurologic Cases, ibid. **22**:99 (July) 1929; Reflex Response Latencies in Manic and Depressive Cases of the Cyclothymic Group and in Cases of Catatonic Stupor of the Schizophrenic Group, ibid. **27**:687 (March) 1932.

lated. Electric currents were supplied both by a standard Harvard coil and by a General Radio beat-frequency oscillator, type 613-A. The coil was used with two dry cells delivering from 0.7 to 1 ampere through the primary circuit. The coil separation was 12 cm., with the secondary coil set at 45 degrees. Approximately 1 volt was supplied to the nerve tissue from the inductorium with the interrupter running. The beat-frequency oscillator was set to deliver alternating currents of relatively pure sine wave form and of various frequencies and intensities. In the experiments on dogs the patellar reflex response latencies were studied, and in the experiments on rats the achilles reflex response latencies. The latencies were determined by the action current technic.[2] All mean reflex response latencies were based on from eight to fifteen records. A difference of 1 σ between two means is considered significant for both the patellar reflex in the dog and the achilles reflex in the rat. In all of the experiments the records were obtained during continuous application of the stimuli. Several minutes were allowed to elapse between the application of one type of stimulus and that of another type of stimulus. In animals in which we were able to record reflexes from both sides, the reflex responses of one side for a given stimulus followed immediately those of the other side for the same stimulus.

By means of the beat-frequency oscillator and a three-stage amplifier activating a vacuum tube voltmeter, the distribution of electric potential through increasing amounts of nerve tissue (brain, spinal cord and sciatic nerve) was measured. The oscillator was set to deliver from 12.5 to 70 millivolts at frequencies of 50, 200, 400, 600, 1,000 and 10,000 cycles.

EXPERIMENTAL DATA

Because each animal presented an individual problem, the data do not lend themselves to condensed tabulation. However, certain reactions occurred generally in all of the animals.

The reflex times (shown in the accompanying table) obtained under the various experimental conditions both in the dogs and in the rats show a notably consistent lengthening of the reflex response latencies during stimulation of any area of the brain.

Only one animal (dog 3) showed any reduction (0.9σ) in the reflex time during stimulation of the brain (right patellar reflex, acid stimulation). The number of animals in each group was too small to permit any definite conclusions as to whether one type of stimulus has more effect than another or as to whether the stimulation of one area has more effect than the corresponding stimulation of another area. The stimuli furnished by electric currents and by acid increased the reflex time more than did those furnished by pressure alone.

For three of the four rats on which the 200 and the 20 cycle currents were used, the amount of current remaining constant (0.6 volt), the 200 cycle current was more effective. It is interesting to note that the exposure of the entire hemisphere in dog 1 acted as a relatively strong stimulus. In the animals for which we

2. Travis, L. E., and Young, C. W.: The Relations of Electromyographically Measured Reflex Times in the Patellar and Achilles Reflexes to Certain Physical Measurements and to Intelligence, J. Gen. Psychol. **3**:374 (July) 1930.

Data for Each Animal

		Reflex Time, σ	
		Mean	Range
Dog 1:	Entire left hemisphere exposed; right patellar reflex time		
	1. Gauze soaked in physiologic solution of sodium chloride covering entire hemisphere	7.3	6.0 - 8.5
	2. Gauze removed	8.4	6.5 - 9.0
	3. Piece of gauze 15 mm. square soaked in acid covering motor area	8.3	8.0 - 8.5
	4. Piece of gauze 15 mm. square soaked in acid covering occipital pole	8.1	6.5 - 9.0
	5. Acid poured over entire hemisphere	7.8	7.5 - 8.0
Dog 2:	Left postrolandic convolution exposed; left and right patellar reflex time		
	Left patellar reflex		
	1. Before operation	6.5	6.5 - 6.5
	2. After operation, no stimulation	6.6	6.5 - 7.0
	3. Silver plate resting on brain	7.4	6.5 - 9.0
	4. Faradic current	7.6	7.0 - 8.0
	5. Acid	7.7	7.0 - 8.0
	Right patellar reflex		
	1. After operation, no stimulation	6.8	6.0 - 7.5
	2. Silver plate resting on brain	7.9	7.5 - 8.0
Dog 3:	Posterior quadrant of left ectosylvian convolution exposed; exposed area, 13 mm. in diameter; left and right patellar reflex time		
	Left patellar reflex		
	1. Exposed area covered with gauze soaked in physiologic solution of sodium chloride	7.7	7.0 - 8.0
	2. Exposed area uncovered	7.1	7.0 - 7.5
	3. Silver plate resting on brain	7.4	7.0 - 8.0
	4. Faradic current	8.7	8.0 - 9.0
	5. Acid	7.5	7.5 - 7.5
	Right patellar reflex		
	1. Exposed area covered with gauze soaked in physiologic solution of sodium chloride	8.2	8.0 - 8.5
	2. Silver plate resting on brain	8.6	8.5 - 9.0
	3. Faradic current	8.4	8.0 - 9.0
	4. Acid	7.3	6.0 - 8.0
Dog 4:	Spinal cord exposed between second and third thoracic vertebrae; left patellar reflex time		
	1. Cord covered with gauze soaked in physiologic solution of sodium chloride	7.6	7.5 - 8.0
	2. Silver plate resting on cord	8.2	7.5 - 9.0
	3. Faradic current	5.4	5.0 - 6.0
	4. Acid	8.2	8.0 - 8.5
Dog 5:	Spinal cord exposed at level of sixth thoracic vertebra; left patellar reflex time		
	1. Without stimulation	8.4	8.0 - 9.0
	2. Generator current, 20 cycles, 0.7 volt	9.2	8.0 -10.0
	3. Generator current, 50 cycles, 0.7 volt	9.2	9.0 - 9.5
	4. Generator current, 200 cycles, 0.7 volt	9.1	8.0 -10.0
	5. Generator current, 500 cycles, 0.7 volt	9.0	8.5 - 9.5
	6. Generator current, 20 cycles, 1.4 volt	9.0	9.0 - 9.0
Rat 1:	Right visual area stimulated; left achilles reflex time		
	1. Without stimulation	7.3	7.0 - 8.0
	2. Silver plate resting on brain	8.6	8.0 - 9.0
	3. Faradic current	9.3	9.0 - 9.5
	4. Acid	9.0	9.0 - 9.0
Rat 2:	Right motor area stimulated; left achilles reflex time		
	1. Without stimulation	7.4	7.0 - 8.0
	2. Silver plate resting on brain	8.2	8.0 - 9.0
	3. Faradic current	8.6	8.0 - 9.0
	4. Acid	8.9	8.5 - 9.5
Rat 3:	Anterior portion of vermis stimulated; left achilles reflex time		
	1. Without stimulation	7.1	6.5 - 8.0
	2. Silver plate resting on brain	7.4	7.0 - 8.0
	3. Faradic current	8.7	8.0 - 9.0
	4. Acid	7.9	7.5 - 8.0

Data for Each Animal—Continued

		Reflex Time, σ	
		Mean	Range
Rat 4:	Anterior tip of right motor area stimulated; left and right achilles reflex time		
	Left achilles reflex		
	1. Without stimulation	7.2	7.0 - 7.5
	2. Faradic current	7.9	7.5 - 8.0
	3. Acid	8.0	7.5 - 9.0
	Right achilles reflex		
	1. Without stimulation	7.0	7.0 - 7.0
	2. Silver plate resting on brain	8.0	8.0 - 8.0
	3. Faradic current	7.9	7.5 - 8.0
	4. Acid	8.0	8.0 - 8.0
Rat 5:	Right motor area stimulated; left achilles reflex time		
	1. Without stimulation	7.0	7.0 - 7.0
	2. Generator current, 20 cycles, 0.6 volt	7.2	6.5 - 8.0
	3. Generator current, 200 cycles, 0.6 volt	8.0	7.5 - 8.5
Rat 6:	Left somesthetic area stimulated; left and right achilles reflex time		
	Left achilles reflex		
	1. Without stimulation	7.0	7.0 - 7.0
	2. Generator current, 20 cycles, 0.6 volt	7.1	7.0 - 7.5
	3. Generator current, 200 cycles, 0.6 volt	8.0	7.5 - 8.5
	Right achilles reflex		
	1. Without stimulation	7.8	7.5 - 8.0
	2. Generator current, 20 cycles, 0.6 volt	7.8	7.5 - 8.0
	3. Generator current, 200 cycles, 0.6 volt	8.5	8.0 - 9.0
Rat 7:	Left visual area stimulated; left and right achilles reflex time		
	Left achilles reflex		
	1. Without stimulation	7.2	7.0 - 7.5
	2. Generator current, 20 cycles, 0.6 volt	7.6	7.5 - 8.0
	3. Generator current, 200 cycles, 0.6 volt	7.9	7.5 - 8.0
	Right achilles reflex		
	1. Without stimulation	7.1	7.0 - 7.5
	2. Generator current, 20 cycles, 0.6 volt	7.1	6.5 - 7.5
	3. Generator current, 200 cycles, 0.6 volt	7.3	7.0 - 7.5
Rat 8:	Left visual area stimulated; left and right achilles reflex time		
	Left achilles reflex		
	1. Without stimulation	7.7	7.0 - 8.0
	2. Generator current, 20 cycles, 0.6 volt	8.1	7.0 - 8.5
	3. Generator current, 200 cycles, 0.6 volt	8.0	7.5 - 8.5
	Right achilles reflex		
	1. Without stimulation	8.1	7.5 - 8.5
	2. Generator current, 20 cycles, 0.6 volt	8.5	8.5 - 8.5
Rat 9:	Cord stimulated at level of sixth thoracic segment; left achilles reflex time		
	1. Without stimulation	7.7	7.5 - 8.0
	2. Silver plate resting on cord	8.2	8.0 - 9.0
	3. Acid	8.5	8.0 - 9.0
Rat 10:	Cord stimulated at level of sixth thoracic segment; left achilles reflex time		
	1. Without stimulation	9.0	8.0 - 9.5
	2. Silver plate resting on cord	9.6	9.0 -10.0
	3. Generator current, 20 cycles, 0.6 volt	9.5	9.0 -10.0
	4. Generator current, 200 cycles, 0.6 volt	10.1	9.5 -11.0
Rat 11:	Cord stimulated at level of seventh thoracic segment; left achilles reflex time		
	1. Cord covered with gauze saturated with physiologic solution of sodium chloride	9.2	8.5 -10.0
	2. Cord uncovered	9.0	8.5 - 9.5
	3. Silver plate resting on cord	7.8	7.0 - 8.0
	4. Generator current, 20 cycles, 0.6 volt	10.0	9.0 -11.0
	5. Generator current, 200 cycles, 0.6 volt	6.1	5.5 - 7.0
Rat 12:	Cord stimulated at level of third thoracic segment; left achilles reflex time		
	1. Without stimulation	8.2	8.0 - 8.5
	2. Generator current, 20 cycles, 0.6 volt	8.5	8.5 - 8.5
	3. Generator current, 200 cycles, 0.6 volt	6.5	5.5 - 8.5
	Cord severed at level of first thoracic segment; stimulated at end of distal segment; left achilles reflex time		
	1. Without stimulation	5.0	5.0 - 5.0
	2. Generator current, 200 cycles, 0.6 volt	7.2	5.0 - 8.5

obtained bilateral reflex latencies (rats 4, 6, 7 and 8 and dogs 2 and 3) there was no evidence that stimulation of one hemisphere affects the reflex time on one side more or less than on the other side.

In both dogs and rats the results obtained from the stimulation of the cord above the segment containing the central connections of the particular reflex arc involved were inconsistent. In dog 4 the faradic current produced the significant reduction of 2.2 σ in the reflex latency. In dog 5 there was a consistent tendency for all stimuli to produce an increase in the reflex time.

Fig. 1.—Typical record of reflex response latency (dog, patellar reflex). Reading from above downward, the first line is the time line (σ), the second is the action current line, and the third is the signal line. The reflex time was read as 9 σ.

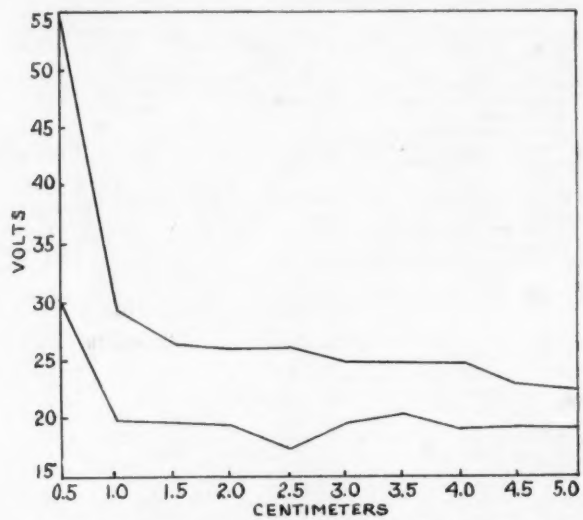

Fig. 2.—Voltage distance curves for the dog's brain. The input current was 400 cycles, 16 millivolts. The source electrodes were placed at the posterior cerebral pole. The search electrodes were placed first 5 mm. anterior to the search electrodes, then 10 mm., then 15 mm., and so on until the anterior cerebral pole was reached. In the upper curve both pairs of electrodes were puncturing the pia-arachnoid while in the lower curve they were resting on the dura.

In rats 9 and 10 there was a lengthening of the reflex time with all stimuli used. In rat 11 there was a striking reduction in reflex time with the mechanical and with the 200 cycle current stimulation. In rat 12 there was a striking reduction in reflex time with the 200 cycle current stimulation. In rat 12, after the cord was severed at the level of the first thoracic segment and the end of the distal segment stimulated by a 200 cycle current, there was a significant increase in

reflex time. It is noteworthy that in rat 12 before the cord was severed the 200 cycle current produced extreme variation in the reflex time during a single series of records. In this animal the latencies varied from 5.5 to 8.5 σ.

Typical curves showing the "spread" of electric stimuli applied to the brain are presented in figure 2. The drop in voltage in both the brain and the sciatic nerve was striking for the first 5 mm. but relatively small and gradual beyond this distance.

COMMENT

In general, the application of direct stimuli to all of the main cerebral fields resulted in lengthening of the reflex response latencies. These results support those of previous experimentation and strengthen further the theory that increased activity of the higher neural levels is associated with decreased activity of the lower neural levels.

Although the electric and acid stimuli increased the reflex time more than did pressure stimuli alone, the biologic effectiveness of the different types of stimuli was not determined experimentally, as the several intensities of those stimuli were not equated.

No convincing explanation occurs either for the fact that the stimulation of one cerebral field did not affect the reflex time more than did the stimulation of another field or for the fact that the stimulation of a cerebral field did not affect the reflex time of one extremity in any different degree than it affected the reflex time of the opposite extremity.

Furthermore, no satisfactory explanation occurs for the fact that in certain instances all the types of stimulation used failed to produce alterations in reflex time. It should be emphasized that the faradic and the 200 cycle current rarely failed to produce significant changes in reflex time. We are unable to interpret why one animal (dog 3, acid stimulation, right patellar reflex) showed a decrease in reflex time during stimulation of the brain.

In both the dog and the rat the results of stimulation of the spinal cord are characterized by frequent inconsistencies. These inconsistencies occur particularly in the direction of numerous shortenings (instead of lengthenings) of the reflex time. Especially do the shortenings occur in response to the faradic and the 200 cycle current. It must be borne in mind, however, that the stimuli were applied to the intact cord, so that in all probability they affected the upper as well as the lower neural centers. Stimulation of the intact cord conceivably affects both the afferent and the efferent arc of both the proximal and the distal segment. In view of these many possibilities for response to a single stimulus, it is impossible to interpret records resulting from such stimulation with scientific accuracy. It may be indicative of summation effects occurring from this combination of possible responses that in the experiments with stimulation of the spinal cord we obtained the greatest

reduction of reflex time (2.2 σ, dog 4, faradic current stimulation) that we have ever obtained from either the dog or the rat following experimental inhibition of the activity of the upper neural centers. The transection of the cord in rat 12 was made in order to eliminate the effects of the stimulation of the proximal segment of the cord. When the cut end of the distal segment of the cord was stimulated by a 200 cycle current, there resulted a consistent notable lengthening of the reflex time (2.2 σ).

A characteristic distribution of potential in all directions from the point of electric stimulation was measured. The potential falls off rapidly with an increase in the distance from the point of application of the stimulus. The spread of the potential refers to the stimulating agent itself and not to a spread of the neural excitatory processes—except, of course, so far as the two might conceivably coincide or even become identical. Either of the two latter possibilities would suggest a significant rôle for voltage decrement as furnishing a restrictive influence on the spread of the stimulus and hence on the possibilities for cerebral response.

SUMMARY

In general, direct stimulation of the cerebral cortex produced a lengthening of the reflex response latency.

Electric stimuli were characterized by a special effectiveness.

Direct stimulation of one part of the cerebral cortex did not affect the reflex time more than did direct stimulation of another part.

Direct stimulation of one cerebral hemisphere affected the reflex time of both extremities to the same extent.

In certain instances, all types of direct stimulation of the cerebral cortex failed to alter the reflex time. In one instance direct chemical stimulation of the cerebral cortex produced a decrease in reflex time.

Direct stimulation applied to the intact spinal cord yielded highly inconsistent results.

Direct stimulation applied to the cut end of the distal segment of a transected cord consistently lengthened the reflex time.

Electric currents of relatively small voltages applied directly to any part of the central nervous system were conducted to circumjacent parts according to a characteristic type of (experimentally measured) potential distribution.

SWELLING OF THE MICROGLIA

REACTION TO INTOXICATION

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AND
BERNARD J. ALPERS, M.D.
PHILADELPHIA

The rôle of the microglia in destructive lesions of the central nervous system is now well established. The production of gitter cells by these elements in response to a destructive process has been demonstrated many times, and their rapidity of response under such conditions is clear. That the microglia are therefore the important phagocytes of the central nervous system cannot be doubted. The rôle which these elements play in the toxic states of the nervous system is not so clear. While they respond quickly to destructive processes, their reaction in toxic conditions is said to be neither rapid nor significant. Indeed, it is stated that these cells are particularly resistant to toxic conditions of any sort.

The purpose of this investigation was to study the reaction of the microglia in toxic conditions of the nervous system in order to establish the rôle which these elements play under these circumstances.

REVIEW OF THE LITERATURE

That the microglia are not completely inert in toxic conditions has been demonstrated by several workers. One of us (B. J. A.[1]) found that while the microglia were slow to react in rabbits intoxicated with urea, and that while formation of gitter cells was never reached, the microglia nevertheless showed a definite and universal reaction to poisoning with urea. This was seen in hypertrophy of the processes and the formation of varicosities and swellings. The hypertrophy in some instances resembled closely formation of rod cells. Similar changes in the microglia were seen in cases of Alzheimer's disease. The changes in the microglia never proceeded to full formation of gitter cells. The

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From the Laboratory of Neuropathology in the Institute of the Pennsylvania Hospital.

1. Alpers, Bernard J.: The Reaction of the Central Nervous System to Experimental Urea Intoxication, Arch. Neurol. & Psychiat. **24**:492 (Sept.) 1930.

possibility of changes in the microglia in toxic conditions has been accepted by del Rio Hortega.[2] On the other hand, Cone [3] expressed the belief that the microglia do not react to toxic or degenerative changes, nor do they react after death.

Changes have been observed in the microglia in experimental lead poisoning. Vizioli [4] found, after injection of lead acetate into rabbits, that the microglia underwent changes of a regressive nature, characterized by a loss of the normal spines and the presence of swellings on the prolongations. The latter seemed to be fewer in number. Vizioli was convinced that the changes were regressive, but admitted the possibility that they might in their early stages be of a progressive nature. Similar changes were observed by Bolsi,[5] who also injected lead acetate into rabbits. He found a rather general hypertrophy of the microglia and giant rod cells in the cornu ammonis. Rabbits given injections of arsenic and mercury showed a more pronounced swelling of the microglia.

The microglia were found to be mobilized rapidly in scarlet fever by Bazgan and Banu,[6] who found hypertrophic forms with fat-bearing granules. The changes described consisted of the presence of shorter processes without spines, with fewer branches and with varicosities and fragmentation. Similar changes were observed by Bazgan and Enachescu [7] in rabbits given injections of rabies virus and *virus fixé*. After from twelve to twenty-four hours there was mild proliferation of the microglia, followed later by hypertrophy of these cells.

Meningitis has been found by Arrigo [8] to cause vacuolization and destruction of the microglia in acute cases, and hyperplasia with rod cell forms in the subacute forms. Radium emanations are responsible

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2. del Rio Hortega, P.: Concepts histogénique morphologique, physiologique et physio-pathologique de la microglie, Rev. neurol **1**:956 (June) 1930.

3. Cone, W. V.: Acute Pathologic Changes in Neuroglia and Microglia, Arch. Neurol & Psychiat. **20**:34 (July) 1928.

4. Vizioli, F.: Microglia e oligodendroglia nella intossicazione sperimentale da plombo, Riv. di pat. nerv. **35**:183, 1930.

5. Bolsi, D.: Ricerche sulla microglia e la oligodendroglia, Riv. di pat. nerv. **37**:1, 1931.

6. Bazgan, I., and Banu, E.: Recherches sur la fonction et le rôle de la microglie dans la scarlatine hypertrophique, Compt. rend. Soc. de biol. **108**:329 (Oct. 16) 1931.

7. Bazgan, I., and Enachescu, D.: Recherches expérimentales sur la microglie, Ann. d'anat. path. **6**:43 (Jan.) 1929.

8. Arrigo, F.: Reazioni microgliali a cause tossinfettive, Riv. di pat. nerv. **35**:256, 1930.

for hypertrophy and an increase in the number of microglia (Bolsi and Conte [9]).

Injections of distilled water into the blood stream of dogs and rabbits or the removal of large quantities of blood is followed by hypertrophic changes in the microglia (Garofeanu [10]).

There is little question, therefore, that the microglia are capable of reacting to toxic conditions of various sorts. In the majority of instances the hypertrophic, and to a lesser degree the hyperplastic, changes noted in the microglia in intoxications have been produced by experiments in which the degree of intoxication has been severe and the dosage of the toxic substance used far from physiologic. In a few cases, however, such changes have been observed in clinical conditions, particularly in chronic degenerative processes, such as senile dementia, dementia praecox, Huntington's chorea, Alzheimer's disease, epilepsy and arteriosclerotic dementia. Similar changes have been found in meningoencephalitis, both purulent and tuberculous (Creutzfeldt and Metz [11]).

MATERIAL AND METHODS

Seventeen male rabbits were used in our experiments. After attempts to produce intoxication in various ways, phosphorated oil (N.F.) was eventually used as the most satisfactory means of causing a uniform toxicity. Histamine was injected into a few animals, but it was found to be excreted so rapidly that it was unsatisfactory as an intoxicant unless used in doses so large as to produce an overwhelming reaction.

Fresh phosphorated oil, in doses of from 0.25 to 0.35 cc., injected intravenously two or three times a week, was found to produce a satisfactory intoxication. Doses of this size could be continued over a long period. Animals were killed at varying intervals of from a few days to several weeks after the injections. Two animals were permitted to live for ten weeks. In two rabbits 1 cc. of the phosphorated oil was introduced into the subdural space, these animals being killed after one and ten weeks, respectively. Injection of the phosphorated oil was always followed by a definite systemic reaction which lasted about twelve hours.

Two animals were killed by bleeding; necropsies were performed immediately after death. In the others, death occurred naturally and necropsy was performed soon after death. Blocks of tissue were fixed in formaldehyde, formaldehyde-ammonium bromide and alcohol, for fat, microglia, astrocyte and cell stains.

EXPERIMENTAL CHANGES IN MICROGLIA

Acute Changes.—In a few animals, which were killed soon after receiving large doses of the phosphorated oil, opportunity was given to study the effects on the microglia in acute toxic states. The first change noted is a loss of spines and

9. Bolsi, D., and Conte, E.: Le reazioni della microglia e della neuroglia alle radiazioni Roentgen e del Radium, Riv. di pat. nerv. **37**:776, 1931.

10. Garofeanu, M.: Réaction de la microglie et de la neuroglie dans l'anémie expérimentale, Compt. rend. Soc. de biol. **97**:1439 (Nov. 25) 1927.

11. Creutzfeldt, H. G., and Metz, A.: The Structure and Activity of the Hortega Cells in Pathological Processes, Ztschr. f. d. ges. Neurol. u. Psychiat. **106**:18 (Dec.) 1926.

side branches. This is followed by the appearance of local swelling along the processes in the form of small "varicosities" or "knobs," noticeable especially at the bifurcations. At the same time there is a swelling of the nucleus, which tends to fill the cell body. The cytoplasm itself may appear swollen and may assume a less intensive stain than normal. In some instances there is a slight widening and rarefaction of the base of the processes. As the changes become more marked the processes appear to retract, becoming thicker and shorter and taking on a gnarled appearance. Portions of the processes may even become frayed and fragmented. The nucleus and cytoplasm meanwhile become more swollen and rare-

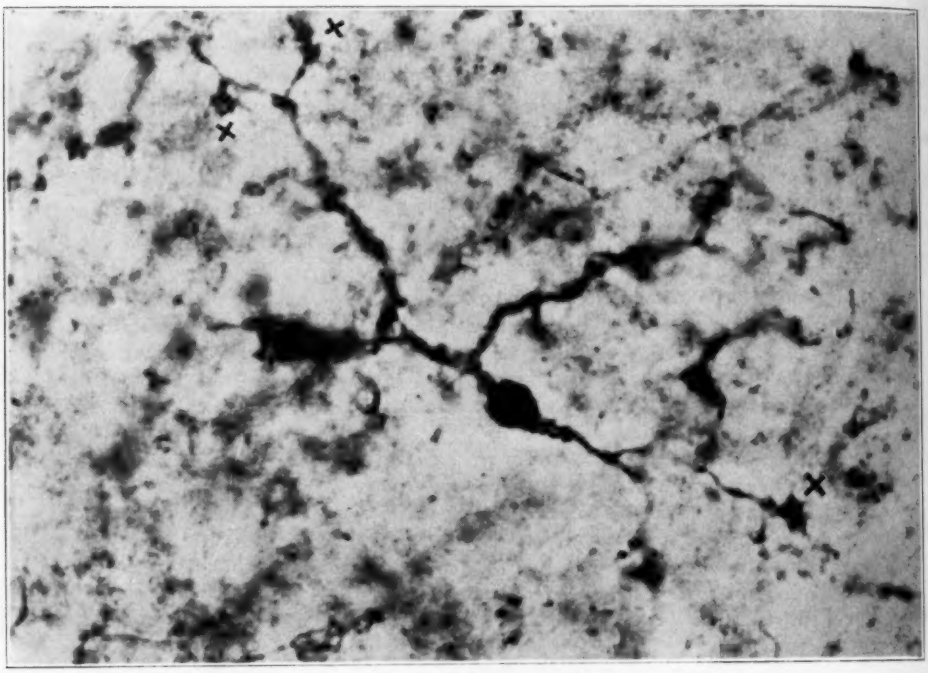

Fig. 1.—A microglia cell, showing numerous varicosities (*X*) along the processes. Silver carbonate stain of del Rio Hortega; reduced from a magnification of × 1,900.

fied, assuming a light gray, granular appearance. Small vacuoles appear in the cytoplasm, the processes become swollen and fragmented, the vacuoles increase and finally the processes are seen as rows of discrete vacuolated nodules.

Chronic Changes.—The changes to be seen in animals which lived for from two weeks to two and one-half months after the injections differ from those in the more acute cases. Here, too, there is in the beginning a diminution in the number of spines and side branches. The processes are thickened and gnarled, with "knobs" along their course, especially at the bifurcations. At the same time there is enlargement of the cell body.

The changes from this point on may be hypertrophic or degenerative. In the hypertrophic group there are enlargement of the nucleus and cytoplasm, which stain more intensely than usual, and thickening of the processes. Not only may the processes retain many of their spines, which are thickened and hypertrophic, but the spines themselves may actually be swollen. The hypertrophic changes in these cells are seen most markedly in the cornu ammonis, where they are normally somewhat larger than microglia in other parts of the brain. In the cornu ammonis these cells may become almost monster-like, assuming typical rod cell forms.

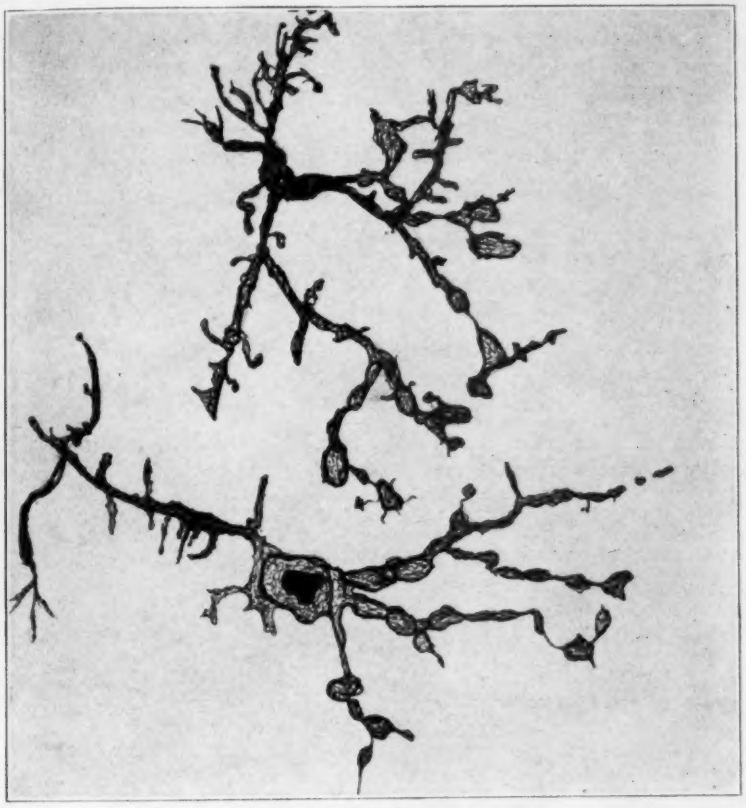

Fig. 2.—Two microglia cells from an animal poisoned with phosphorus, showing the loss of branches, the hypertrophy of the remaining processes and the vacuolation and the varicosities.

The degenerative changes are characterized by the occurrence of "knobs" or "varicosities" along the branches. These swellings become globular and rarefied, and finally completely vacuolated. Eventually the processes themselves become vacuolated, frayed and fragmented, often appearing only as a series of large clear globules or vacuoles connected by thin filaments. The cytoplasm becomes vacuolated and swollen, leaving a clear wide space between the cell membrane and a thin portion of vacuolated cytoplasm which encircles the nucleus. The cell body

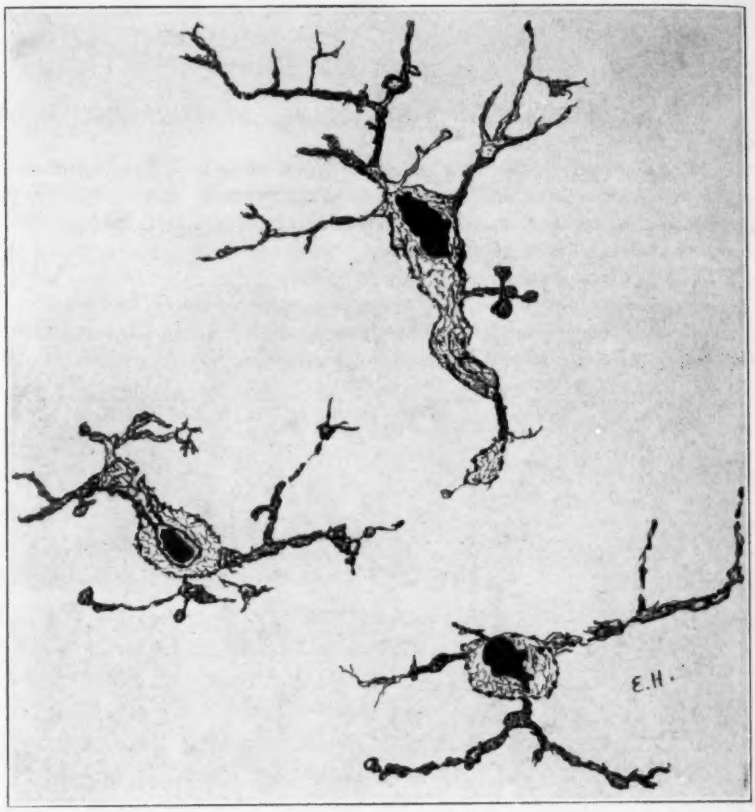

Fig. 3.—A group of microglia cells, showing varicosities, loss of spines and side branches and swelling of the cytoplasm.

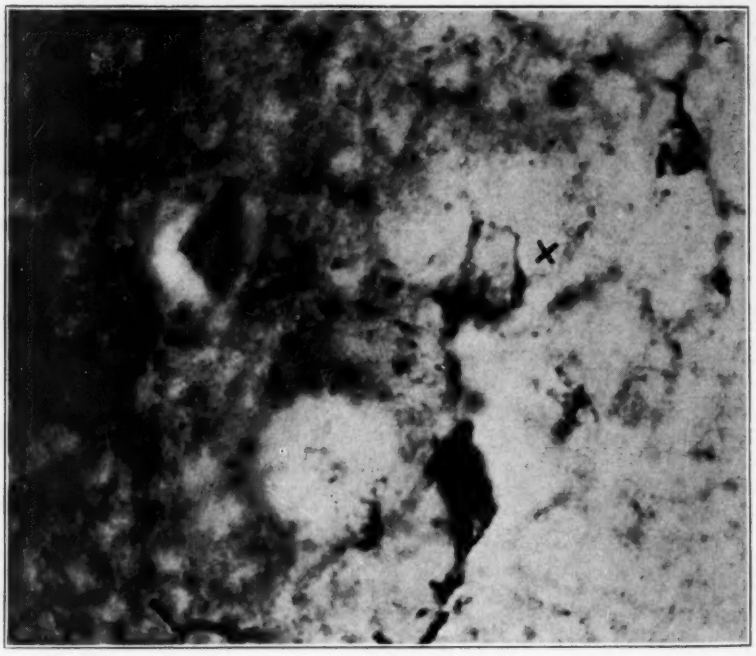

Fig. 4.—A very large vacuolated process of a swollen microglia cell (*X*). Silver carbonate stain of del Rio Hortega; reduced from a magnification of × 1,900.

seems to become ballooned out, a process which extends into the bases of the processes.

In no instance were gitter cells found, except in the region of small embolic foci that were present in a few specimens. Fat could not be demonstrated in any of the microglia cells, whether hypertrophic or degenerated.

A careful study of the nerve cells showed no changes. The Nissl substance was intact, and the ganglion cells were no different from those seen in control animals. It is difficult, however, to be absolutely dogmatic about this since not a great deal is known about the normal cerebral cortex of the rabbit. In a careful comparison with control cortices of the rabbit, however, no deviations from the normal were found.

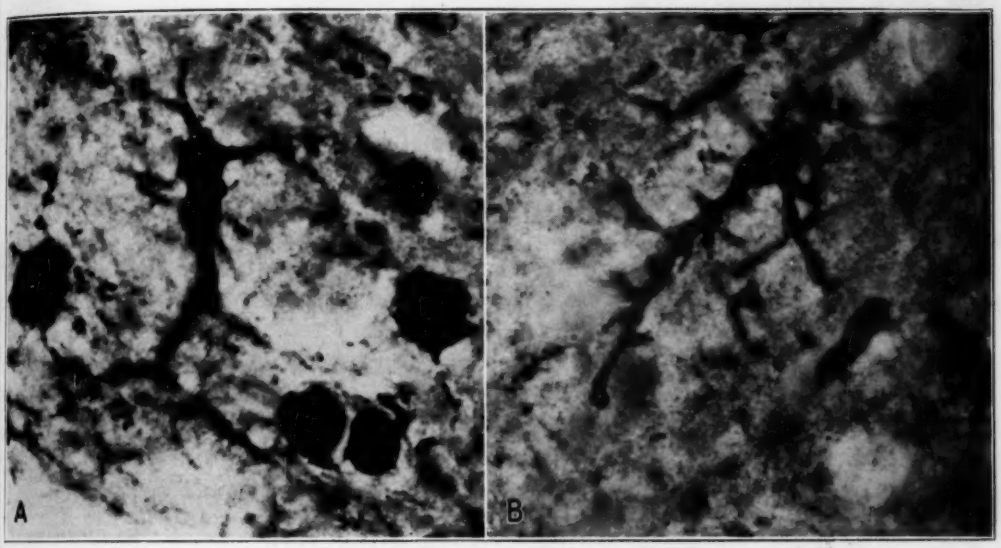

Fig. 5.—Two very large, hypertrophied microglia cells. Note the thickening of the remaining process and the loss of the finer branches and spines. In *A* there is a tendency to formation of rod cells. Clubbing is evident in *B*. Silver carbonate stain of del Rio Hortega.

CHANGES OF THE MICROGLIA IN TOXIC CONDITIONS IN MAN

We have observed changes in the microglia similar to those produced by experimental phosphorus poisoning in a few conditions in man. In acute poliomyelitis we have found swelling and vacuolation of the microglia in the cerebral cortex, of a more pronounced degree than that seen under experimental conditions. Similar changes were found in the cerebrum of monkeys given injections of poliomyelitis virus. Marked swelling of the microglia was noted in the cortex in a case in which an intraventricular injection of a phosphorus-containing compound had been made in order to outline the ventricles. The swelling and vacuolation were severe in this case, both in the microglia and in the oligodendroglia. Hypertrophic changes in the microglia, with the formation of varicosities, have been

observed by one of us (B. J. A.) in Alzheimer's disease. Swelling of marked degree has been found in a case of subacute bacterial endocarditis with cerebral foci.

SUMMARY AND COMMENT

Toxic Reaction of Microglia.—There can be little question that the microglia react to toxic substances introduced into the blood stream, or, furthermore, that injection of toxic substances into the subdural space or directly into the cerebrum likewise causes definite changes in the microglia cells. A rather uniform type of reaction in these cells has been observed after the injection of heavy metals, such as lead and mercury, after the introduction of arsenic and phosphorus and the viruses of rabies, poliomyelitis and scarlet fever, after bleeding and in other conditions. In man similar changes have been observed in poliomyelitis, subacute bacterial endocarditis with cerebral foci, Alzheimer's disease, meningitis, meningo-encephalitis and a variety of other conditions, particularly in the arteriosclerotic and senile groups.

The nature of the change has been uniform. In the overwhelmingly fatal intoxications the microglia have reacted by localized hypertrophy, seen in the varicosities along the processes as well as by swelling, vacuolation and fragmentation of the cytoplasm and processes. In the less severely toxic cases, on the other hand, there has been a rather pronounced hypertrophy of the microglia, sometimes with the production of what appear to resemble rod cells, especially in the region of the cornu ammonis. At the same time vacuolation of the cytoplasm and processes, with swelling and globule formation, and eventual fragmentation were seen to occur. Whether the latter is the result of degeneration of the hypertrophic forms is hard to say, but it would seem so from the appearance of the histologic sections. The occurrence of rod cell forms confirms the finding of del Rio Hortega,[12] who found such forms in experimental urea intoxication. Furthermore, their presence under such conditions is suggestive, indicating possibly the stimulus required to evoke their universal response in such diseases as dementia paralytica. Their presence was noted only in cases in which subtoxic doses were administered over a long period of time. In brains containing these forms, moreover, it was easy to detect transitional stages from the more severe changes observed in the overwhelmingly toxic cases. Repeated subtoxic doses of an intoxicant seem to be capable, therefore, of evoking a microglial response in which the cells may assume a rod cell form.

It has been asserted that the microglia are sluggish in their response to intoxicants and that, furthermore, when they do react it is only in the

12. del Rio Hortega, P., in Penfield, W.: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 2, p. 528.

presence of a severe intoxication. Del Rio Hortega has recently reiterated this point of view. He asserted that previous destruction of the nerve structures is required for the intervention of the microglia, and such is the case only in very acute intoxications. It is true that the conditions necessary for the production of the changes seen in the microglia consist on the whole of a severe toxicity. Profound intoxications elicit the reactions noted more readily than milder ones, but our experiments demonstrate that similar changes may be produced by small dosages continued over a long period. In other words, similar changes can be produced by intoxications which are not overwhelming and not severely acute but are rather chronic in nature. Furthermore, our experiments suggest that the microglia are not unresponsive to intoxications, and that they do react to toxic stimuli. Their response seems to be more pronounced in the human brain than under experimental conditions, for in cases of poliomyelitis and subacute bacterial endocarditis we were able to find marked evidence of microglial swelling, more pronounced even than the experimentally produced changes.

The possibility that the type of change observed in these conditions may be due to technical errors has been expressed by Villaverde,[13] who found no changes in the microglia in experimental lead poisoning which he thought could not be explained by technical difficulties. No such possibility enters into our cases in which necropsies were performed immediately after killing the animals, and in which fixation was equally prompt. That the changes we have observed may take place post mortem is possible, but that they are not of this nature in our cases is equally certain. Acute swelling of oligodendroglia takes place after death, but it is well established also that it is a charteristic reaction in many toxic-infectious conditions during life.

Nature of the Changes.—It is important to know whether the response of the microglia to intoxicants is regressive or whether it represents an effort on the part of these cells to assume phagocytic or reparative activity. Fat could not be demonstrated in any of the cells, even in those with large vacuoles. Furthermore, in no instance could gitter cells be demonstrated. The presence of rod cell forms is suggestive of a reparative function, but these forms were in no sense universal enough to assume such a factor as definitely shown. Rod cells have been shown to be possibly a mildly phagocytic form of microglia, and in this sense the cell forms noted may be assuming phagocytic power.

On the whole, however, one is impressed with the fact that the changes observed in the microglia in response to intoxicants are largely,

13. Villaverde, J. M.: Lésions du cerveau causées par les sels de plomb, Trav. du lab. de recherches biol. de l'Univ. de Madrid **26**:9, 1928-1929.

if not entirely, regressive. The hypertrophy and the varicosities, vacuolation and dissolution of the processes suggest such a hypothesis. These forms probably have no function in removing the intoxicant from the blood stream. They may, however, serve as sacrifices to the action of the poison, preventing its penetration to the nerve cells. When formation of rod cells takes place, the cells may serve some supportive or obscure reparative function.

CONCLUSION

1. Seventeen rabbits were given injections of phosphorated oil, usually intravenously, but twice subdurally.

2. Changes were observed in the microglial elements.

3. The changes observed were probably chiefly regressive, but may have been in part reparative.

CONVULSIONS OF UNDETERMINED ETIOLOGY

STUDIES OF THE BLOOD SUGAR

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LOS ANGELES

The concept of so-called idiopathic epilepsy is rapidly changing. For ages, recurrent attacks of convulsions associated with unconsciousness were thought to constitute a primary disease. By degrees, as newly discovered organic causes of such seizures became definitely established and recognized, the large group of cases in which a diagnosis of "idiopathic epilepsy" would formerly have been made shrank steadily. At the present time over fifty separate conditions are recognized as "causes" of convulsions. If, in the study of a case, a cause is discovered, the convulsions are considered to be a symptom of the primary disease. A case of idiopathic epilepsy is an undiagnosed case; the term is pernicious in that it masquerades as a diagnosis. It has no more *raison d'être* than such terms as idiopathic fever, idiopathic hematuria or idiopathic paralysis. However, the term is still in common use and is here applied to epileptiform phenomena of undetermined origin.

Hypoglycemia (blood sugar below 0.075 per cent) is of common occurrence. In certain people it is closely associated with hypotension and with relative overactivity of the parasympathetic nervous system in general. This has been emphasized by Munch-Petersen and Schou.[1] Probably one sixth of all persons tend to have a low percentage of blood sugar. The tall, thin, asthenic type of person is almost certain to show hypoglycemic characteristics, but many others also fall into the category of hypoglycemic and hypotensive persons.

TECHNIC

In determining whether or not a person has a tendency toward hypoglycemic periods it is not sufficient to examine a single specimen of blood after fasting for twelve hours. I have found such a single determination to be almost useless. A dextrose tolerance test should be done; for the sake of uniformity and for purposes of comparison it should be conducted approximately as follows: After a twelve hour fast (about 8 a. m.), a specimen of blood is taken. The patient then drinks a solution of dextrose (from 30 to 50 per cent) which is flavored with lemon juice to make it more palatable. The dextrose should equal 1.75 Gm. per kilogram of body weight. Specimens of blood are then taken one-half, one, two, three and four

1. Munch-Petersen, C. J., and Schou, H. I.: Investigations into Sugar-Metabolism in Epileptics, Especially the Sugar Threshold in Adrenalin- and Glucose-Hyperglycemia, Acta psychiat. et neurol. **6**:545, 1931.

hours after ingestion. Blood sugar determinations are made (in the curves here reported by the method of Folin and Wu) in terms of milligram per hundred cubic centimeters of whole blood. During the test one should note clinically whether the patient manifests evidences of a "hypoglycemic reaction," i. e., weakness, extreme hunger, tremor, sweating, tachycardia or even coma.

It is difficult to overemphasize the importance of the six determinations, because, while the low point usually appears at the third hour, it may come at the fourth. Occasionally it appears at the second hour. It is disappointing, after the test, to see the curve still on the decline, since this means that the lowest point probably had not been reached.

It is the failure to carry out the test over a sufficiently long period of time that has led to such divergent opinions concerning the value of the concentration of blood sugar in patients with epilepsy. Schwab,[2] in a general study of blood sugar in nervous diseases, found hypoglycemia in some cases, but did not continue the curve beyond the third hour. Daly, Pryde and Walker [3] thought that there might be a relationship to seizures, but found the results inconclusive. Incidentally, they also concluded, correctly, that hypoglycemia is not peculiar to epilepsy. An apparently crushing blow to the idea that epilepsy is dependent on hypoglycemia was given by Lennox, O'Connor and Bellinger,[4] who concluded after elaborate, extensive and careful work that the blood sugar played only a passive rôle. However, they conducted tolerance tests for only two hours and consequently did not regularly discover the low points. Munch-Petersen and Chou [1] thought that there was perhaps a displacement toward lower values of about 10 mg. in determinations during fasting of blood sugar in epileptic persons, but agreed with Lennox that this was not of definite pathologic importance.

Greisheimer [5] showed that nervous irritability in decerebrate dogs rose with the lowering of the blood sugar concentration and fell with its rise. In a series of epileptic patients Josephs [6] found that a short fast, especially after vigorous exercise, brought on convulsions. In only one of his cases was excessive hypoglycemia found. These attacks were all associated with fever and vomiting. Ross and Josephs [7] presented

2. Schwab, S. I.: Diagnostic Value of Blood Sugar Curves in Neurology, Arch. Neurol. & Psychiat. **8**:401 (Oct.) 1922.

3. Daly, I. De B.; Pryde, J., and Walker, J.: The Blood Sugar in Cases of Epilepsy, Brit. M. J. **1**:232 (Feb. 9) 1924.

4. Lennox, W. G.; O'Connor, M., and Bellinger, M.: Chemical Changes in the Blood During Fasting in the Human Subject, Arch. Int. Med. **38**:533 (Nov.) 1926. Lennox, W. G., and Bellinger, M.: Studies of Metabolism in Epilepsy: The Blood Sugar Curve, Arch. Neurol. & Psychiat. **18**:395 (Sept.) 1927.

5. Greisheimer, E. M.: Irritability and Blood Sugar, Am. J. Physiol. **72**:213, 1925.

6. Josephs, Hugh: Fasting as a Cause of Convulsion, Am. J. Dis. Child. **31**: 169 (Feb.) 1926.

7. Ross, S. G., and Josephs, H. W.: Observations on the Metabolism of Recurrent Vomiting, Am. J. Dis. Child. **28**:447 (Oct.) 1924.

case records which convinced them that hypoglycemia might be the determining factor in at least some seizures in cases of idiopathic epilepsy. Griffith [8] concluded that, at least in some instances in children, a causative relationship of hypoglycemia to convulsions might be reasonably assumed. Sudden spontaneous fluctuations in the concentration of the blood sugar have been reported by Ashe, Mosenthal and Ginsberg.[9]

Conclusive evidence that hypoglycemia may, in the presence of other factors, be the final provocative agent or be an underlying factor in the causation of convulsions has come forth with the knowledge that the pancreas produces insulin (Banting and Best); that tumors of the islands of Langerhans occur with resultant overproduction of insulin and convulsive seizures;[10] that removal of these tumors relieves the patient from the attacks;[11] that functional dysinsulinism may occur, and that relief from convulsive seizures may be produced by raising the blood sugar concentration.[12] Removal of part of the pancreas as a remedy for hyperinsulinism has also been performed with some degree of success.[13]

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8. Griffith, J. P. C.: Hypoglycemia and the Convulsions of Early Life, J. A. M. A. **93**:1526 (Nov. 16) 1929.

9. Ashe, B. I.; Mosenthal, H. O., and Ginsburg, G.: Hypoglycemia: With and Without Insulin; With and Without Symptoms, J. Lab. & Clin. Med. **13**:109 (Nov.) 1927.

10. (*a*) Wilder, R. M.; Allen, F. M.; Power, M. H., and Robertson, E. H.: Carcinoma of the Islands of the Pancreas, J. A. M. A. **89**:348 (July 30) 1927. (*b*) Howland, G.; Campbell, W. R.; Maltby, R. J., and Robinson, W. L.: Dysinsulinism, ibid. **93**:674 (Aug. 31) 1927. (*c*) Thalheimer, W., and Murphy, F. D.: Carcinoma of the Islands of the Pancreas, ibid. **91**:89 (July 14) 1928. (*d*) McClenahan, W. U., and Norris, G. W.: Adenoma of the Islands of Langerhans with Associated Hypoglycemia, Am. J. M. Sc. **177**:93 (Jan.) 1929. (*e*) Allan, F. N.; Boeck, W. C., and Judd, E. S.: The Surgical Treatment of Hyperinsulinism, J. A. M. A. **94**:1116 (April 12) 1930. (*f*) Allen, F. N.: Hyperinsulinism, Arch. Int. Med. **44**:65 (July) 1929. (*g*) Carr, A. D.; Parker, R.; Grave, E.; Fisher, A. D., and Larrimore, J. W.: Hyperinsulinism from B-Cell Adenoma of the Pancreas: Operation and Cure, J. A. M. A. **96**:1363 (April 25) 1931.

11. Womack, N. A.; Gnagi, W., Jr., and Graham, E. A.: Adenoma of the Islands of Langerhans with Hypoglycemia, J. A. M. A. **97**:831 (Sept. 19) 1931. Howland, Campbell, Malthby and Robinson.[10b] Allan, Boeck and Judd.[10e] Carr, Parker, Grave, Fisher and Larrimore.[10g]

12. Nielsen, J. M., and Eggleston, E. L.: Functional Dysinsulinism with Epileptiform Seizures, J. A. M. A. **94**:860 (March 22) 1930. Harris, Seale: Epilepsy and Narcolepsy Associated with Hyperinsulinism. Report of Three Cases of Epilepsy and of One Case of Narcolepsy Cured Clinically by Partial Resection of the Body and Tail of the Pancreas, ibid. **100**:321 (Feb. 4) 1933. Love, J.: Dysinsulinism: A Discussion and Case Report, ibid. **100**:814 (March 18) 1933.

13. Finney, J. M. T., and Finney, J. M. T., Jr.: Resection of the Pancreas, Am. J. Surg. **88**:584 (Sept.) 1928. Holman, Emil, quoted by Allen, Boeck and Judd.[10e] Allen.[10f]

However, hypoglycemia is not always of pancreatic origin. Gammon and Tenery [14] outlined its possible origins: (1) hyperfunction of islands of a normal number; (2) hypertrophy of the islands; (3) hyposupra-

Dextrose Tolerance in Serial Cases of Idiopathic Epilepsy *

Blood Sugar in Milligrams per Hundred Cubic Centimeters of Whole Blood

Case	Fasting	Half Hour	First Hour	Second Hour	Third Hour	Fourth Hour	Low Point
1	94	110	93	101	54	74	54
2	93	143	114	83	64	73	64
3	78	106	78	63	68	75	63
4	97	...	160	142	69	..	69
5	80	109	67	75	73	65	65
6	86	86	82	80	80	..	80
7	72	80	105	100	80	..	72
8	71	83	54	54	60	..	54
9	80	81	97	62	75	..	62
10	83	90	105	60	71	..	60
11	83	83	86	74	57	..	57
12	85	122	100	97	72	89	72
13	83	69	87	125	74	54	54
14	97	..	148	59	..	..	59
15	61	102	80	78	58	57	57
16	..	108	95	85	83	61	61
17	85	125	138	75	58	..	58
18	80	100	105	74	62	..	62
19	66	...	76	95	..	..	66
20	74	...	69	66	..	..	66
21	76	66	69	71	76	..	66
22	103	133	109	113	108	71	71
23	72	114	48	70	70	60	48
24	85	105	111	95	95	57	57
25	82	106	102	93	86	70	70
26	59	92	67	65	61	61	59
27	82	99	98	79	79	..	79
28	86	172	158	92	78	62	62
29	93	...	155	51	..	..	51
30	81	83	63	60	63	..	60
31	96	132	72	102	114	98	72
32	75	105	102	100	121	54	54
33	80	125	100	86	64	..	64
34	93	100	62	90	80	..	62
35	69	153	133	90	55	..	55
36	69	58	76	81	64	..	64
37	71	76	66	64	57	76	57
38	103	132	166	99	70	81	70
39	80	100	106	86	64	86	64
40	64	152	117	91	58	..	58
41	91	166	200	143	142	133	91
42	80	100	105	69	74	80	69
43	70	153	137	54	57	69	54
44	76	105	88	95	60	72	60
45	86	137	89	89	85	66	66
46	76	125	86	74	74	80	74
47	100	154	107	89	72	87	72
48	81	142	69	102	60	90	60
49	..	108	95	85	83	61	61
50	86	143	105	87	75	54	54
51	153	250	181	174	166	166	153
52	77	...	80	54	..	71	54
53	80	142	125	95	83	..	80
54	80	90	76	62	54	..	54
55	79	135	114	91	82	..	79
56	95	110	133	81	74	68	68
57	58	119	103	93	46	51	46
58	99	89	93	84	93	94	84
Total	4,634	5,968	5,905	4,947	4,001	2,566	3,777

* In cases 5, 13, 15, 16, 22, 24, 25, 28, 32, 50 and 56 the lowest point was reached at the fourth hour.

renalism; (4) hypothyroidism; (5) hypopituitarism; (6) interference with the storage of glycogen in the depots of the body, and (7) loss of

14. Gammon, G. D., and Tenery, W. C.: Hypoglycemia, Arch. Int. Med. **47**: 829 (June) 1921.

dextrose from the body. Relative to the last-named origin renal glycuresis has been established as an entity [15] and is of considerable importance, when present, in preventing a normal rise in blood sugar.

ORIGINAL INVESTIGATIONS

For about two years all available patients with idiopathic epilepsy have been subjected to a dextrose tolerance test when consent could be obtained and the work carried out. Tests conducted by the capillary method are not comparable with those done by examining the venous blood; hence, the cases in which the former method was followed are not included in this report. In some instances the work was done at laboratories of the patients' choice; incomplete tests were common, and most reports of these have not been used. Tests continued for three hours usually gave a low reading at some points. Many of the tests were conducted on patients at the clinic at the White Memorial Hospital. The curves obtained are shown in the table, with the inclusion of three cases previously published as occurring in cases of dysinsulinism. The cases have been unselected except that careful examination failed to reveal a cause for the seizures. Cases have been included even though during the test an occasional specimen was lost or was not taken.

COMMENT

In evaluating the tests it should be stated that the blood sugar level is always lower at some point during a dextrose tolerance test than at any time following a mixed meal. The curves, therefore, do not represent what occurs daily in these patients; they are used for comparative purposes only. This immediately raises the question: Do these patients usually have attacks during the dextrose tolerance test? They do not. Hypoglycemia alone, unless extreme (0.040 per cent), probably is not sufficient to cause an attack. Other factors are probably one or all of the following: hydration, alkalosis, depletion of glycogen, hyposuprarenalism or a temporary severe imbalance in the vegetative nervous system.

The dextrose tolerance curves presented show several types. Occasionally a curve like that of a diabetic person, or one within the normal range, is found. Otherwise, an excessively low point is obtained or a flat curve is found in which a constantly low blood sugar occurs (which, however, is not low enough as a rule to be called hypoglycemia). Curves have been considered normal if the figures fell between the two presented here:

Fasting	Half Hour	First Hour	Second Hour	Third Hour	Fourth Hour
110	170	140	120	100	110
80	120	110	90	80	90

Yet many persons of a vagotonic constitution have readings below these levels and are not considered ill. One must use discretion in

15. Jones, L.: Hypoglycemia, M. Clin. North America **8**:949 (Nov.) 1924.

determining whether a given curve indicates a pathologic condition. One must be still more careful in the application of determinations. Munch-Petersen [1] believed that a tendency to lower readings existed in persons with epilepsy, but saw no practical application of the fact.

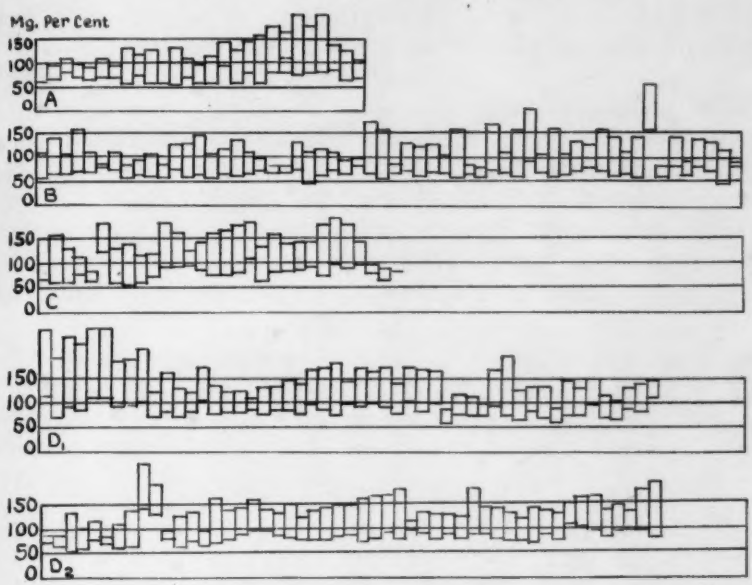
Because of the indefiniteness of what is to be considered a normal curve, because of the individual factors in determinations of blood sugar and because the curves illustrated were obtained from various laboratories in which the technic certainly varies to some extent (the time allowed after the drawing of the blood before determination of the sugar is made, the manufacture of standard, anticoagulant utilized, etc.), another approach was taken to the same problem. Through Dr. O. B. Pratt, in charge of the laboratory of the College of Medical Evangelists, access was obtained to a series of consecutive dextrose tolerance curves, all of which had been obtained in the outpatient department of the teaching clinic. The same technic had been used throughout. Only curves from cases in which a final diagnosis had been reached were utilized, and the records were not followed back farther than to the point in time at which three hour tests had become routine. In a great many of the cases a specimen had also been taken at the fourth hour but, as many of these were of epileptic patients and a sufficient number of specimens of normal persons could not be obtained, all specimens taken at the fourth hour are omitted in this presentation.

I grouped the curves into four classes, those of (1) diabetic patients, (2) epileptic patients, (3) neurotic persons (all cases of psychoneurosis, "vegetative imbalance," except when epilepsy coexisted, "endocrine states" and migraine) and (4) the normal controls (obviously not normal persons, as the patients had come to the clinic for medical attention; but even when selecting a series of normal people one does not ordinarily make a complete physical and laboratory examination to ascertain that such persons are absolutely normal; the final diagnoses in these cases included nothing which, so far as I know, affected definitely and regularly the blood sugar level). A few of the curves are suggestive of diabetes and a few are certainly typical of vagotonia, but when one is selecting cases for a study of blood sugar, one must not use the blood sugar readings in arriving at a diagnosis. The final diagnoses were accepted as they were made. The curves of diabetic persons were at once discarded as having no application in the present problem.

The number of curves utilized were those of: normals, or controls, 102; epileptic patients, in whom no cause for the epilepsy had been determined, 50, and neurotic persons, 30, making a total of 182 curves. To conserve space, voluminous tables showing all the curves have been condensed into block graphs presenting only the highest and the lowest points of each curve. These are shown in the chart. In preparing the block graphs the cases common to both series of epileptic patients were

subtracted from *A* and are shown only in *B*, so that all duplication has been avoided.

The chart shows that, whereas a few of the normal controls (D_1 and D_2) show a blood sugar approaching the low level of 50 mg., many of the epileptic patients (*A* and *B*) show a low level.[16] *C* shows the neurotic persons to be midway between those with epilepsy and the controls. Furthermore, in the blocks of the normal persons there are many above the 150 level (35 of 102), while in the diagrams of the epileptic persons (*A* and *B*) there are relatively few (16 of 85).



Graphs of the high and low points in the concentration of blood sugar in each reported case: *A*, graph of the epileptic patients of the control series; *B*, graph of the epileptic patients of the series here reported (those common to both *A* and *B* are given only in *B;* many approach the 50 line); *C*, graph of neurotic persons of the control series; D_1, D_2, graphs of the normal controls; (few approach the 50 line).

A composite of the tables of all the control series (the averages of the entire number of each group of cases and for each of the five specimens obtained), to which has been added the average of the low points in the curves, is shown in the following tabulation.

Type of Case	Fasting	Half Hour	First Hour	Second Hour	Third Hour	Low Point
Controls.....(102)	88	137	129	103	87	79
Neurotic persons.... (30)	85	127	123	103	81	77
Epileptic patients... (50)	80	119	104	87	76	70

16. The average low point for 102 normal persons was 79 and for the 50 epileptic patients of the same series it was 70.

It was found that if the specimens taken at the fourth hour had been calculated, a still lower low point would have been obtained.

The composite blood sugar curves from the series of 58 cases from personal experience are compared with a similar curve from the 50 cases of the control series (23 cases of which are common to both series) in the following tabulation.

	Fasting	Half Hour	First Hour	Second Hour	Third Hour	Fourth Hour	Low Point
Personal cases....	83	115	102	85	75	75	65
Control series....	80	119	104	87	76	..	70

The lower figure for the low point in my own series is due to inclusion of the specimen taken in the fourth hour in that series.

From the foregoing it is clear that as a rule the blood sugar in neurotic persons, probably because of a vagotonic preponderance, is slightly lower than in the controls. Otherwise the two types of curves are practically alike. But in the group with epilepsy the blood sugar is about 10 per cent below that of the controls. In addition, the drop in blood sugar in the second half hour is definitely to a lower level than in the control group.

Since the tendency to hypoglycemia is present in about 90 per cent of the cases of idiopathic epilepsy, that finding may serve to differentiate certain cases of convulsions. However, it greatly complicates the differentiation between hyperinsulinism and idiopathic epilepsy, actually tending to show that these states merge imperceptibly.

SUMMARY OF CONCLUSIONS

The term idiopathic epilepsy means to me an undiagnosed syndrome of epilepsy. A series of 58 consecutive dextrose tolerance curves in such cases is presented, and shows that periodic or constant low blood sugar occurs in about 90 per cent of epileptic persons during a dextrose tolerance test. A series of 182 consecutive dextrose tolerance curves is also analyzed to determine whether, as patients with all sorts of trouble appear, those with epilepsy, as a class, show lower readings. The syndrome, idiopathic epilepsy, seems nearly confined to persons with a tendency to hypoglycemia, but except in rare instances hypoglycemia itself is not sufficient to cause an epileptic attack. Other factors necessary are probably hydration, alkalosis, depletion of glycogen, hyposuprarenalism or imbalance in the vegetative nervous system. I agree with Munch-Petersen and Schou that the hypoglycemia rather serves to indicate the type of person with which one is dealing. However, hyperinsulinism cannot be detected and separated from idiopathic epilepsy by the determination of blood sugar during fasting. It cannot be differentiated even by a dextrose tolerance test. Clinical data of other sorts are necessary.

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SPECIAL ARTICLES

THOUGHT IN SCHIZOPHRENIA

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Beyond doubt the most significant development in psychology has been the recent tendency to bring together investigations in various fields in order to discover the common principles involved in those investigations. Especially is this true of psychopathology and genetic or child psychology. They have developed independently, and only occasionally heretofore have the results obtained been brought together for comparison.

Meanwhile more and more investigators think that psychologic laws are the same no matter where they are observed. As an example of the growing integration of the various fields of psychologic investigation and the growing feeling of the unity of psychologic laws in spite of the variety of their manifestation, one may refer to the comparative study of the splitting of thought, the phenomena of hypobulia [1] in psychopathology, and the phenomenon of syncretic thinking in child psychology. In hypobulia there are phenomena which were formerly considered a result of schizophrenia or hysteria; in the light of more thorough investigation, however, they now appear to be in reality stages in the normal organization of consciousness as a necessary ontogenetic step in the development of normal personality. Such observations are doubtless widely true: The phenomena of developing thought in the adolescent child are evidently in general closely related to certain aspects of pathologic thinking.

For the past five years Vigotsky, together with Professor Luria, has been doing extremely interesting work on the psychology of schizophrenia, utilizing the experimental technic of the Gestalt psychology. The article was written at my request over three years ago, and since then a great deal more work has been done.

Dr. Charles Trueblood of Brown University edited the translation (translator's note).

1. "The hypobulic type of will is the ontogenetic and phylogenetic lower stage of the purposive will" (Kretschmer, E.: Hysteria, Washington, D. C., Nervous and Mental Disease Publishing Company, 1926).

There is, furthermore, a growing tendency to investigate psychologic processes by observation and comparison of their various courses of development, this procedure being used as a means of arriving at the laws determining the characteristics of such processes. I have found such an approach extremely useful in clinical and experimental work. Whereas previously the bringing together of genetic psychology and psychopathology consisted merely in a comparison of the conclusions reached, I have attempted to introduce the comparative method of study into my own experimental work from the beginning. It did not take long to find out that many problems, so conceived, appeared in a totally different light.

There is an old attempt to connect the psychology of adolescence with certain symptoms of schizophrenia. This connection was implied in the term "dementia praecox," and has given stimulus for a large number of studies of the adolescent child and for the comparison of the mental life of the child with that of patients with schizophrenia. Kretschmer, in Germany, and Blonsky,[2] in Russia, insisted that there is a connection between the two. They based their opinion on the fact that at times it is impossible to differentiate between a stormy period of sex adjustment in adolescence and incipient schizophrenia. My investigations, on which I shall comment later in the article and which give rise to certain ideas about the nature of psychologic processes in schizophrenia, lead me to quite different conclusions. The pivotal point in my comparative analysis has been the process of formation of concept as observed in the child and in the patient with schizophrenia.

SCOPE OF INVESTIGATION

My investigations have been twofold. They have embraced the development of thought in children up to the age of puberty, on the one hand, and the deterioration of thought in schizophrenia on the other. The conclusions have likewise been twofold. I have found that the most important development of thought in adolescence is the change from "complex" [3] types of thinking to conceptual types of thinking—a change which not only revolutionizes the intellectual processes but determines the dynamic structure of the personality, i. e., the consciousness of the self and of the environment. I have also found, conversely, that the

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2. Blonsky, P.: Pedologia, Moscow, Rabotnik Prosveschenia, 1926.

3. By complex thinking Vigotsky signifies not the usual meaning of the term "complex" in psychopathology but a type of simple elementary generalization found in the thought processes of a child, a primitive man or a psychotic patient. This type of thinking can perhaps be expressed in terms of English psychology as associative thinking or "group thinking," meaning by "group" a unity whose members are different, i. e., a type of thinking in which groups of different elements are related to each other (Domarus).

most important deterioration of thought occurring in schizophrenia is a disturbance, an impairment, in the function of formation of concept. The fragmentation and the breaking of that part of the psyche which is involved in the process of formation of concepts is just as characteristic of schizophrenia as the development of the function of formation of concepts is characteristic of adolescence. Hence it is obvious that both in schizophrenia and adolescence certain external similarities can be found, especially in the transition from complex [4] or associative to conceptual thinking. When both are approached in a formal, static way during the transitional stages a large number of points in common can be found. But by using a more dynamic method of approach it will be seen that the psychologic processes in schizophrenia and in adolescence have a converse relationship to each other and that they are connected more by differences than by similarities. This is true, if for no other reason, because in adolescence one is dealing with phenomena of growth and development, while in schizophrenia one is dealing with the disintegration and decay of psychic life. Such principles obtain for the general mental processes of the person, but they are especially applicable in the function of formation of concepts. By studying this function, one becomes convinced that the psychology of adolescence gives a key for the understanding of schizophrenia, and conversely that schizophrenic thought helps one to understand the psychology of adolescence. In both, the most important thing is the proper understanding of the function of formation of concepts.

METHOD

My experiments consisted in offering the patient a situation which required the formation of artificial concepts. This was accomplished by giving the patient what appeared in the beginning to be meaningless words chosen at random. The formation of the concepts had to be based on specially selected and connected elements. Thus, in the series of experiments the patients had to learn to associate meaningless syllables with certain definite concepts, as for example, "bik," meaning large and small, "lag," meaning large and tall, etc. The patient, that is, was confronted with the problem of the formation of a new concept, which he would not meet anywhere else except in the setting of a laboratory experiment.

It should be stated parenthetically that this method of experimental formation of concepts by means of specially selected words has a long history into which I shall not enter at present. It suffices to say that the method has been used a great deal by Ach [4] and his students. My

4. Ach, N.: Ueber die Begriffsbildung, Untersuchungen zur Psychologie und Philosophie, Bamberg, Ach, 1921, vol. 3.

methods of investigation were based on principles advanced by Ach, but as I used them for altogether different purposes they had to be considerably modified.

With the methods developed by my collaborator, L. S. Sacharov,[5] we were able to observe the impairment of the faculty of formation of concepts, not only when the disturbance of thought was quite apparent, but also in the cases in which no formal disorder of thought could be demonstrated. The important fact here is not that the patient with schizophrenia, confronted with the experimental problem, is not able to solve it, but that, in the attempt to solve it, he exhibits characteristic and significant forms of thought.

OBSERVATIONS

Not counting refusals and half-hearted cooperation in the experiments, in all cases in which the results were definite and clearcut we observed certain characteristic forms of association which resulted in the formation of certain kinds of ideas taking the place of concepts. We could adduce a large variety of these forms of association, but what we believe essential at the moment is the description of the common characteristic of such associative processes. I shall enumerate the most frequent associative structures encountered: (1) collective thinking, in which various objects are grouped together as if they formed a collection composed of different objects united to each other by certain relationships—such as a collection of things or objects of various colors or various forms; (2) chain complex thinking; (3) associative complex thinking; (4) pseudocomplex chain thinking. The last three will be explained later. All of them imply a whole constituted of organically united parts, the difference between such associations and concepts being that in the associations the union is concrete and mechanical, whereas in the concept there is a general and abstract principle on the basis of which the conceptual association is formed. A complex is best likened to a big family in which are grouped, under the same family name, a large number of altogether different people. A patient with schizophrenia looks on the stimulus word as a family name for a group of objects on a basis of physical proximity, concrete similarity of certain parts or some other nonabstract relationship to each other. A typical example would be the so-called chain associations in patients with schizophrenia. The patient responds to a stimulus word denoting a certain object by naming another object similar in only one trait, then naming a third object chosen on account of some similarity to the second object, then in similar fashion adding a fourth to the third, etc.

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5. Sacharov, L. S.: The Methods for Study of Concepts, Psychologia **3**:1, 1930.

The result is a number of quite heterogeneous objects very remotely connected with each other. The associative chain is built up in such a relationship and in such a manner that there is a connection between separate links but with no single principle uniting all the links. Thus, in my experiments the subject has to select a group of objects, all of which have a common name, being guided in the principle of grouping by a sample shown to him. The example may consist of a small blue triangle, then a large round green figure, then a green parallelogram, etc. (the chain color complex); or he may be shown the same triangle and may choose another triangle which is quite different from the first in color and size (associative complex). There arises thus a joining of various objects resembling a large family in which the tie is of a most heterogeneous character, degree and principle. Such a method of association is common in children before adolescence. In spite of all the differences in the process of thought in the child and in the patient with schizophrenia, there is a fundamental similarity in the most essential features. Thus in persons with schizophrenia thought is really regressive.

COMMENT

The impairment of formation of concept leads back to complex thinking, and although the concepts which were formed previously are used well and quite automatically, the formation of new concepts becomes extremely difficult. There is an important conclusion to be derived from such observations. Comparison of thought in persons with schizophrenia with the various genetic stages of complex thought establishes a psychologic criterion, a means of evaluating the degree of splitting and regression in the patient with schizophrenia. The disintegration of concepts and the regression to the concrete, factual, complex forms of thought have been observed by other investigators without appreciation of the genetic factors involved in the differentiation between complex and abstract thinking. This failure finds its expression in the fact that the comparison of disordered thinking with phylogenetically earlier forms of thought is usually made on the basis of negative rather than positive criteria, merely on the basis of the absence of concepts in thinking. This comparison, based on a negative criterion, is wrong because it treats as approximately equivalent forms of thinking which from the positive side have nothing in common with each other—which are, in fact, separated by many millions of years in genetic development. The example to be cited will explain this.

Some authors compare the complex thinking of persons with schizophrenia with the thinking of primitive people, with thought in dreams, and finally with intellectual processes in lower animals, especially with the process of thought in spiders as has been shown by Volkelt. As reported by Volkelt, the spider goes through accurate movements when

trying to get its prey from the web into the nest but becomes lost when the same prey is removed from the web—that is from the total complex situation to which the spider is accustomed—and placed directly in the spider's nest. The selective consciousness of a spider does not so much perceive isolated sensations as perceive total conditioned emotional situations. In all these the transition to associative thinking is represented as a step toward visual, pictorial thinking. Although a trend is undoubtedly in evidence, all these comparisons suffer by disregarding the degrees of the governing psychogenetic development. Between abstract thinking in the form of concepts and thought as it is exhibited by the spider, there are a great many developmental steps, each one differing from the other no less than the associative thought of the patients with schizophrenia differs from the thought of a normal person.

And just as it is not admissible to make a genetic comparison of thought as it occurs in dreams with thought as it occurs in primitive man or in spiders, simply because such forms of thought are all below the stage of conceptual thought, neither has one the right to assume that the thought of the patient with schizophrenia immediately drops into the abyss of millions of years or needs for its understanding analogies with the spider, which does not recognize its prey after the prey has been removed from the web and placed in the nest.

My observations show that complex thought observed in patients with schizophrenia is the nearest step to conceptual thought and immediately precedes it genetically. There is some similarity, then, although by no means an identity, between the thought of the patient with schizophrenia and the thought of a child. The one common basis which permits direct comparison of the two different types of thinking is that the process of thought of the child and that of a patient with schizophrenia in the initial stages of the disease are merely steps in the genetic development of thought; that is, they represent the step immediately preceding the stage of formation of concept and cannot be compared to the process of thought of the spider from which they are separated by millions of years of development. One knows that even in adulthood there remains a tendency to complex thinking in certain fields. A superficial examination will not reveal the transition from one mode of thinking into another unless special methods of investigation are employed.

A second important conclusion to be derived from the experiment relates to the fact that in schizophrenia there is a destruction of the psychologic systems which lie at the basis of concepts. Expressing the same idea differently, it can be said that early in schizophrenia the meanings of words become changed. These changes are sometimes difficult to observe unless one uses special methods, but they can be demonstrated. The way to understand this phenomenon lies in the study

of thought in the child. A child thinks differently from an adult; consequently the words for him also have different connotations in their psychologic structure. The question naturally arises—If the words have different meanings how do a child and an adult understand each other? As an example I may cite the paradoxical fact established by Piaget that children of the same age and degree of development do not understand each other as well as they do adults. Yet the thinking of adults is governed by laws quite different from those determining the thinking of children. This, it will be seen, involves the problem that I described at the beginning of this article. If, I said, the meaning of words begins to change early in the course of the schizophrenic process, how does that fact remain unobserved, and how is it possible for the normal person and the person with schizophrenia to understand each other?

The answer to such questions, as indicated by my investigations, lies in the fact that "complexes" may and sometimes do coincide with concepts in their reference to objects, but not necessarily in their meanings. When one speaks of Napoleon as the victor at Jena and the loser at Waterloo, the two phrases coincide in their reference to Napoleon, but are widely different in their meanings. When a concept and a complex thus refer to the same object, the complex may be spoken of as a pseudoconcept. Pseudoconcepts, which are basic elements in the thought of a child, may coincide with the concepts of adults, but this does not mean that they necessarily coincide in other particulars. When a child says "house" or "dog," he may be speaking of the same objects as the adult, but he thinks about them in a different way. He groups and combines them in a way quite different from that of the adult.

The fact that in its reference to objects the speech of a child coincides with the language of the adult can be explained by the development of speech in children. Speech in a child does not develop freely and spontaneously; the child does not create words and their meanings. He finds them both ready made in his environment, and he acquires something that has been prepared for him. In his environment certain names are definitely attached to certain objects. Each object has its distinctive name, and the child, acquiring these names, groups them by the only method he knows, i. e., by associations. The association consists of objects not chosen freely by the child, but is made on the basis of existing connections and relationships of the objects with each other, relations in part previously established by the adult. As soon as this external pressure is removed, the associations of the child and the concepts of the adult begin to differ, not only in their connotations but also in their relationships to objects. My study of the thinking of deaf-mutes shows that they have associative thinking and that they even resort to earlier

forms of thought—the syncretic forms of making connections. Thus, in the mimic language of the deaf-mute the gesture denoting teeth may also mean "white," "stone" and "talk," depending on the whole sentence. The additional gestures, such as pointing to the upper lip, or indicating rejection or pointing, make possible the differentiation of various meanings which are all united on the basis of the associative complex, of which I have already given examples. But because their mimic speech develops without the fixed system of rigid limitations associated with verbal speech, their associations do not coincide in relationship to objects with the concepts of normal adults. This same situation exists in schizophrenia. The words of the patient with schizophrenia coincide with ours in their object relationships but not in their meanings.

PROCESS OF THOUGHT IN SCHIZOPHRENIA

Two influences determine such a phenomenon. The first is that (with the exception of neologisms) the patient with schizophrenia uses in his speech the system of fixed names which he learned in childhood. When the disintegration begins, he reverts to complexes, in the place of concepts, not freely, but as predetermined by his prior attachment of certain names to certain situations and objects. A table is a table for us as well as for a patient with schizophrenia, but we think about it differently. He puts all the various tables into a complex, and the word table is merely a familiar name for this association. We use a general concept, and the name is merely carried as a symbol of this concept. In other words, he has in his possession a ready-made system of words standing in definite relationship to the objects they denote. Consequently, since he does not see the principle forming the basis of this association, his association is invariably a pseudoconcept.

The other influence involved in the fact that the words of the patient with schizophrenia coincide with those of the normal person in their object reference but not in their meanings arises from the way in which conceptual thinking develops. I have said that a school child goes through a stage of complex thinking as a period in his development immediately preceding conceptual thinking. Consequently, in ontogenesis, complexes precede concepts and actually form the inner layer or the older substructure beneath the new layers of concepts, if one utilizes Kretschmer's graphic expression for older and newer forms of thought. There is reason to believe that the development of concepts, like the appearance of other higher psychologic functions, is accomplished by the formation of new layers over the old ones, with the preservation of the older layer of thought in a subordinate function. This law, which was recently discovered in the development of the central nervous system, holds true also for the development of various psychologic functions, motor as well as central. Kretschmer has shown

that hypobulia, i. e., the early stage in certain motor discharges, is preserved in all the activities of the organism associated with the discharge of volitional impulses. Hypobulia is preserved in a latent, subordinate rôle, and occasionally it is uncovered and expresses itself independently when the higher processes of the will are impaired or disturbed. Something like this must be taking place in schizophrenia. Associations, as a primitive form of thought, are retained as a substructure in the development of the higher forms of thinking, but they are uncovered and begin to act independently in accordance with their own laws when the whole personality, for some reason, is disturbed. There is reason to believe that complex thought is not a specific product of schizophrenia, but merely a cropping out of the older forms of thought, which are always present in a latent form in the psyche of the patient but which become apparent only when the higher intellectual processes become disturbed by illness. The regression to earlier forms of thought is observed also in other diseases in which there is interference with conceptual thinking. The process of thinking then becomes strikingly similar to thought in schizophrenia, and this probably accounts for the schizophrenic reaction in the course of physical illnesses. The other proof that these are earlier forms of thought can be found in the fact that associative thinking is latent in all of us and comes to the surface in connection with sudden emotional shocks and in a setting of fatigue, sleep and dreams. There is nothing impossible, then, in the assumption that regression of patients with schizophrenia to complex thinking is merely a reversion to earlier forms of thought. Each one of us carries schizophrenia in a latent form, i. e., in the mechanisms of thought which when uncovered become the central figure in the drama of schizophrenic thought. Thus, the history of the development of thought ought to be used as a means of reaching an understanding of the peculiarities of complex thinking in schizophrenia.

ALTERATIONS IN THE MEANING OF WORDS

Whatever may be its cause, and paradoxical as it may appear, the fact is nevertheless fairly well established that the meanings of words become pathologically altered in schizophrenia, though such alterations do not become apparent for a long time. Complexes replacing concepts in thought in schizophrenia nevertheless coincide in their object relationships with the concepts they replace. They are then pseudoconcepts, but the whole transition to the more primitive forms of thought is not apparent because the patient retains his capacity for verbal intercourse, even though words do not have the same meaning for him as they have for us. As a concrete illustration I may cite my experimental investigations as to the degree to which patients with schizophrenia at the same stage of the disease, and with the same type of thinking, under-

stand each other as compared with the degree of mutual understanding exhibited by a patient with schizophrenia and a normal person. As might be expected, the experiments indicated a better mutual understanding between patients with schizophrenia and normal persons than between schizophrenic persons. An analogous situation is seen in children, who understand adults better than they do each other. The solution of this problem is presented later.

An important question, which to me is a central question of schizophrenia, arises in this connection. If it is really true, as I assert, that in schizophrenia there is disintegration of concepts with changes in the meanings of words, even though this is not apparent on the surface, there must be some proofs that these phenomena actually take place in schizophrenia. The proof is simple. If words have different meanings for a patient with schizophrenia from those which they have for us, then this difference must express itself functionally, i. e., in the behavior of the patients. Even if a complex may outwardly resemble a concept, it nevertheless has its own laws of function. Just as the associative thinking of a child expresses itself in various ways, so must the thought of a patient with schizophrenia reveal its distinguishing characteristic when subjected to a test, i. e., in actual behavior. This was the principle of my experiments, and I found that in actual function these associations reveal the changes in the meanings of words which I postulated previously.

From many methods I have selected the test dealing with capacity for metaphorical expression, i. e., the transference of terms originally denoting one thing to the expression of others. (A ship plows the sea.) I first used this test in cases of aphasia associated with loss of memory, in which may also be seen disturbances both of categorical thinking (Gelb and Goldstein) and of conceptual thought. (In this connection it may be noted that the disturbances of categorical thinking, which Gelb and Goldstein found as a cardinal symptom in amnesic aphasia, were also found by them in a patient who exhibited amnesia for various colors. When asked to match colors, this patient instead of matching objects according to the color designated, would match them according to size, or according to value in brightness of the paint, and only occasionally according to color, thus manifesting the previously described complex type of thinking.) I found in my patients an analogous and marked disturbance in the capacity both for using words in metaphorical senses and for understanding words so used. They could not grasp the meanings of the simplest words unless they were used in a direct and literal sense. Nor could they cope with the test of Piaget, which requires the subject to match a specified proverb with another of similar meaning. To my surprise such failures occurred in spite of an apparent preservation of speech and of other intellectual

functions. I later discovered, however, that Kurt Schneider had also found disturbances in the capacity to understand words used in metaphorical senses to be a frequent characteristic of schizophrenia. Most remarkable was the fact that I found disturbances in the understanding of words figuratively used, even when there was no apparent disturbance of intellectual life in general. This difficulty became very obvious when special words or concepts were used. While the normal mind has no difficulty in using given words metaphorically or figuratively, the same problem presents insurmountable difficulty for the patient with schizophrenia in spite of the fact that he has retained from childhood the habit of using figures of speech, proverbs, etc. Thus, many of my patients have no difficulty in seeing the wider ramifications and generalities when they are given the Russian proverb, "If you go slowly you get further in the end," but they could not give a general meaning when the Russian translation of a French proverb, "When the cat is away the mice will play," was given. This they interpreted in its narrow sense, and they could only see literally that mice play when the cat is away. They could not, that is, see, in a situation concretely described, meanings other and more abstract than those directly signified by the particular words used in describing it. This fact serves as an important differentiation between the visual, symbolic thinking of dreams and the metaphorical, symbolic thinking based on concepts. The identification of one with the other is without any solid, psychologic basis.

FORMATION OF NEW CONCEPTS

I found also another fact illustrating disturbances of meaning in words used by patients with schizophrenia. My experiments did not stop at the stage of development of experimental concepts. I studied the manner in which these new concepts expressed themselves. I included them as a part of association tests in which the responses were carefully traced out. The subjects were asked to make judgments which included the old as well as the newly formed concepts, and were encouraged to widen the application of the newly formed concepts and to carry them over from the laboratory into every-day life. In other words, I wanted to trace as fully as possible the course of the newly formed concepts in the thinking of the patients. Without going too much into detail, I may state that there was found a latent disintegration of concepts. I found also that the pseudoconcepts which took the place of true concepts were quite different from them in behavior and expression. As an example of pseudoconcepts I may take the example of the concept of causality in a child. As the reader will remember, a child begins quite early to use words denoting causal relations, such as the word "because," although, as Piaget has shown, the meaning given by the child to these words differs altogether from that given by an adult.

A child will connect causally the most inconsequential ideas, a fact which led Piaget to speak of a certain stage in the development of a child as a precausality stage. One must have special methods to demonstrate such pseudoconcepts because superficially they may resemble true concepts in their external appearance. Pseudoconcepts are wolves in sheep's clothing. They are associations which look like concepts. Anybody who works with them finds out quickly how they disturb the forms of conceptual thinking. In order to demonstrate this, however, one must consider other psychologic functions. As an example of the more remote consequences resulting from the disturbance of the function of concept formation, I may refer to experiments with perceptions and with affective responses in schizophrenia. A study of the perceptions of a patient with schizophrenia indicates that for such a patient various common perceptual objects easily lose their common perceptual characteristics. Slight variations in light or in the position of the object bring out in the patient responses similar to those of normal persons to the meaningless ink blots of the Rorschach test. Just as normal persons see in such ink blots people, landscapes, faces, fairies and what not, so does the patient with schizophrenia, in his perception of objects, attach to them the most extraordinary meanings if there is the slightest change in their customary appearance. The key to the understanding of the phenomenon lies in genetic psychology, which teaches that categorical perceptions are achieved through a complicated process, in which percepts and concepts are coordinated into new forms of visual thinking, the percepts playing therein a subordinate and dependent rôle. As an example of such fusion of conception and perception in the narrow sense of the word I may refer to illusions, in which one cannot separate the meaning from the object (white shadow-ghost). It is also known from experimental psychology that it is impossible under normal conditions to get absolute perceptions without associating with them meanings, understandings and apperceptions.

This is why it is so hard to get perception in pure culture, and why objects cannot serve all of us as ink blots serve us in the Rorschach test. Perception is an integral part of visual thinking and is intimately connected with the concepts which go with it. This is why every perception is really an apperception. But this is not true for complex thought. With the disintegration of concepts and their regression to more primitive forms of thought, the whole relationship between perception and apperception becomes altered in a manner which is typical of schizophrenia. Such a change is closely akin to the phenomena which appear in the affective life of patients with schizophrenia. The significant factors here are not the emotional dulness and the disappearance of the richness and variety of emotional expression, but the separation of these emotional expressions from the concepts with which they are

closely associated. These facts, of course, are well known clinically. My contribution lies in the demonstration of the fact that disturbance of emotional life is only part of the wider and more fundamental disturbance, i. e., a disturbance in the field of concept formation. My postulation is that the intellectual disturbance, as well as the disturbances in the fields of perceptions, emotions and other psychologic functions, are in direct causal relationship with the disturbance of the functions of formation of concepts. This hypothesis is based on the results of the developmental study of the individual, i. e., on ontogenetic data.

GENERAL COMMENT

A study of the development of psychologic functions in childhood through adolescence affords an opportunity to observe the connection between development of the capacity for formation of concepts and the development of personality. In adolescence one finds a fundamental regrouping of these various functions, a complete change of their interrelationships, leading to the appearance of totally different psychologic systems of a much higher order and complexity. A disintegration of these new systems, a splitting of those higher functions, is what is found in schizophrenia.

But the investigation brings out still other conclusions. The capacity for formation of concepts is really the third of three stages in the intellectual growth of the child. The first involves the development of ideas of physical causality. The second consists in secondary changes in other psychologic functions. The third, intimately connected with the formation of concepts, also involves the development of personality and a world outlook, i. e., the cognition of one's self and one's environment. The appearance of a formed personality with a world outlook in adolescence is the result of the highest development of intellect in that period of life. The process has been discussed elsewhere in my work on the psychology of adolescence. Observing the disturbance in the perception of self and the environment by the patient with schizophrenia, I cannot but believe that there is some involvement of the third stage in the development of personality associated with the functions of the formation of concepts. And truly, a perception of the self and the outside world is intimately connected with the concepts by means of which they are represented. One knows that the child's concepts of himself and his environment are quite different from those of an adult. One knows how changed are the perceptions of self and the environment in dreams, and it is fair to suppose that the changes in personality and changes in perception of the outside world observed in schizophrenia are caused by the slumping of intellect from the conceptual level to the level of associations.

True enough, this is only a hypothesis, but it is a tempting hypothesis, not only because it takes into consideration the developmental facts of those functions which are strongly affected in schizophrenia but also because it allows one to reduce the data to a common denominator and to study schizophrenia in the light of the psychologic development of personality.

There is one misunderstanding which invariably appears in any discussion of schizophrenia, and which I should like here to clear up. Utilizing the function of the formation of concepts as a starting point in investigation and finding also that it is the psychologic center or nucleus of the whole drama of the disease, one yet sees that it has nothing to do with the etiology of schizophrenia. Disturbances in the function of concept formation are the immediate result of schizophrenia but not its cause. I am not at all inclined to treat schizophrenia as a psychogenic disorder. Whatever may be the organic cause of the disease, however, psychology has a right to study the phenomena associated with the changes in personality from a psychologic point of view. Disintegration of personality follows certain psychologic laws, even though the direct causes of this process may not be psychologic in nature.

Moreover, the clinical and physiologic observations form a bridge to psychologic speculations. I refer particularly to clinical observations which lead to the conclusion that at the basis of schizophrenia there is a loss of psychic energy. Jung was the first to draw the parallel between dreams and schizophrenia. He put it beautifully, that if a man could walk and talk in his dreams his total behavior would be in no way different from that of a patient with schizophrenia. The asthenic habitus as a constitutional factor has been emphasized by many authors. I had an opportunity recently to study schizophrenia in children in a setting of marked fatigue and sleep. One of my patients was observed to drop off to sleep frequently. He was asleep most of the afternoon, and in the acute stage of the illness the tendency to fall asleep was most marked. I feel that there must be some germ of truth in the old clinical observation comparing stupors and sleep. Although sleep and schizophrenia are not identical, yet they have some points in common. Lately this view found expression in Pavlov's paper on "The Excursion of a Physiologist into the Field of Psychiatry," in which he stated the belief that the most probable physiologic cause of schizophrenia is the overdevelopment of the process of inner inhibitions, which are also overdeveloped in hypnosis and sleep.[6] Some time ago Pavlov thought that cortical inhibitions and sleep were identical; now he believes that inner inhibitions and schizophrenia have a good deal in common. Of course,

6. Kasanin, J.: Pavlov's Theory of Schizophrenia, Arch. Neurol. & Psychiat. **28**:210 (July) 1932.

it is a fascinating theory. The thing which interests me in this theory is the fact that it bridges the gap between the psychologic hypothesis and the physiologic data in schizophrenia. If one recalls that the biologic function and purpose of inner inhibitions, including sleep, consist in cessation of contacts with the outside world, it becomes clear that autism, withdrawal and shutting off one's self from reality are direct results of the special state of the central nervous system of patients with schizophrenia. The loss of contact with the outside world assumes a biologic significance. It is not a result of schizophrenia but an expression of the protective forces of the organism reacting with inner inhibitions to the weakness of the central nervous system. If this is so, and there seems to be every reason to believe that it is a fact, important conclusions may be drawn. All higher psychologic functions, including speech and conceptual thinking, are of social origin. They arise as a means of rendering mutual aid, and gradually they become a part of the person's every-day behavior. It is significant that in dreams there is a cessation of contacts with that social self which forms the foundation of the normal personality. This apparently becomes also the cause of impairment of intellect in the field of concepts; the other symptoms of schizophrenia, as I have shown, all spring from this source. At any rate, my experimental data, interpreted in the light of genetic psychology, allow one to formulate certain thories which I have here presented.

THE DOMAIN OF NEUROPSYCHIATRY AND THE TRAINING OF THE NEUROPSYCHIATRIST

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In the great progress of medicine and surgery during the last half century the development of special fields has been one of the outstanding features. Medicine and surgery have branched out into a number of special activities which are still attached to the parent stem by tradition and practice. Such specialties are built around certain organs and systems of the body, certain diseases and even a specialized therapeutic technic. This concentration of effort has resulted in great progress, but it has tended to narrow the point of view so that what has been gained in depth has been lost in breadth. Today, however, there is a tendency both in teaching and in practice to seek a broader outlook and to avoid the pitfalls of an early and too narrow specialization.

Of all the specialties, that of the nervous system is the most important and the most difficult to master, because of its intricacy, the wide range of its activities and the unusual complexity of its structure. It is the master tissue of the body and the integrating mechanism which regulates all its activities. It is the seat of mental processes which are the highest function of the organism, and therefore it touches every field of medicine. The nervous system is the domain of neuropsychiatry. It includes all those diseases and disorders of function which Jelliffe and White, in their textbook, "Neurology and Psychiatry," assign to the physicochemical, the sensorimotor and the psychic or symbolic systems of the organism. The first system is concerned with visceral neurology or the neurology of metabolism, the second with the neurology of sensation and motion and the third with reactions at the psychic level.

All of these systems, physiologic and psychologic, represent the various stages in the evolution of the central nervous system of man. They are all sensorimotor, and their various reflex activities form a continuous chain, from the simplest twitching of a muscle fiber to the highest realm of thought.

As Hughlings Jackson said, nearly fifty years ago:

> If the doctrine of evolution be true, all nervous centers must be of sensorimotor constitution. A priori, it seems reasonable to suppose that, if the highest centers have the same composition as the lower, being, like the lower, made up

of cells and fibers, they have also the same constitution. It would be marvelous if, at a certain level, whether we call it one of evolution or not, there was a sudden change into centers of a different kind of constitution. Is it not enough difference that the highest centers of the nervous system are greatly more complicated than the lower? Some years ago I asked the question: of what substance can the organ of mind be composed, unless of processes representing movements and impressions?

Therefore, the various reflexes of the visceral and sensorimotor systems and the reactions of the psychic system are of the same fundamental nature, and differ only in the complexity of their manifestations.

HISTORIC BACKGROUND OF NEUROLOGY AND PSYCHIATRY

The history of neurology and psychiatry shows clearly the common origin and close association of those two disciplines.

Neurology.—This field is peculiarly the child of internal medicine, and made its appearance as a special field in the early part of the nineteenth century. The first systematic treatise on the subject was by Moritz Heinrich Romberg, whose "Lehrbuch der Nervenkrankheiten" was published in 1840. The great neurologists of this period and up to the beginning of the present century sprang from internal medicine. Charcot and Duchenne in France, Hughlings Jackson and Gowers in England, Romberg and Nikolaus Friedreich in Germany, Weir Mitchell and Mills in this country were all products of the medical wards.

With the further development of neurology as a special field close relations with general medicine were always maintained. Neurologic consultants were appointed to general and special hospitals in which the neuropsychiatric material of those days was to be found. It was only later that special wards and hospitals for neurologic cases made their appearance, and in this country up to 1900, with few exceptions, the general ward was the field of action of the neurologist. Here were encountered organic and functional disorders of every kind, the psychoneuroses, the minor psychoses and many of the major psychoses not requiring commitment. During these years great advances were made in both the organic and the functional disorders of the nervous system; some idea of the progress attained is shown in the great textbooks of Gowers and Oppenheim and in a host of important monographs of that period.

Advances were made in knowledge of the finer anatomy and histology of the nervous system and its functions, in the recognition, localization and interpretation of the pathologic processes of the various nervous disorders and last, but by no means least, in the sphere of what was then termed the functional disorders of the nervous system. The relation of the neurologist to this large and important group of nervous disorders

is peculiarly close, both historically and by the exigencies of practice. And I think it may be said that the neurologist has consistently made most of the major contributions in this field.

It was in the Charcot clinic, where organic neurology made such great progress, that the psychogenic origin of hysteria was conceived and elaborated, and where Pierre Janet laid the foundation for his epochal work in the psychoneuroses. The contributions to the nature and treatment of the psychoneuroses by later representatives of the French school are too well known to require mention, but such names as Dejerine and Babinski stand out for original contributions in both the organic and the psychic fields.

It was in the London Hospital that Hughlings Jackson developed his great doctrine of physiologic levels which has played so important a rôle in clarifying the conception of the function of the central nervous system, including the psychic level. And above all it was Sigmund Freud, a neurologist of Vienna, who conceived and developed the elaborate structure of psychoanalysis which has revolutionized so many of the ideas of psychology and psychotherapy.

In this country the work of George M. Beard, who developed the concept of neurasthenia which bears his name, and of Weir Mitchell, who described and perfected the rest cure which represented a great therapeutic advance in its time, and Morton Prince's brilliant researches in abnormal psychology may be singled out for special mention, as indicating the important relationship which neurology has always had with the problems of psychologic medicine.

The masterly contributions of these men would not appear to sustain the assumption advanced by some American psychiatrists that practice in organic neurology interposes a barrier to advancement in the psychic realm. One might even suggest that practice in organic neurology acts as a stabilizing factor and tends to direct psychic investigations along sane and orderly channels.

Psychiatry.—The historic background of psychiatry is one of the most interesting in the field of medicine. Like neurology it was an outgrowth of general medicine.

The activity of early psychiatrists, up to very recent times, was largely confined to the hospitals for the insane, and their first efforts were in the direction of ameliorating and humanizing the living conditions and care of these unfortunate patients.

The work of Pinel and Esquirol in France, of William and Hack Tuke and Connolly in England, of Fricke and Griesinger in Germany and of Benjamin Rush and Kirkbride in this country, in advancing this problem is among the great practical accomplishments of medicine.

As soon as better living conditions prevailed in institutions for the mentally afflicted a more scientific psychiatry began to emerge.

Improved methods of treatment and a more orderly classification of mental disease began to emerge. Important works on mental disorders by Esquirol, Hack Tuke and Benjamin Rush appeared, and the first formal textbook on psychiatry was published by Wilhelm Griesinger, in 1845. Like Rush, whose noted treatise, "Diseases of the Mind," appeared in 1812, Griesinger was a product of internal medicine, and made many important contributions to the control of infectious diseases.

In 1818 Heinroth described *die Verrücktheit* (paranoia). Calmeil's celebrated researches in dementia paralytica were reported in 1826. Jean Pierre Falret described "circular insanity" in 1854. The hebephrenia of Kahlbaum and Hecker was described in 1871, and catatonia in 1874.

The writings of Esquirol and Morel in France, Meynert, Wernicke, Krafft-Ebing and Westphal in Germany, and Hack Tuke, Maudsley and Sir Thomas Clouston in England are among the classics of their day.

Some of the most notable contributions to the anatomy and pathology of the central nervous system have been made by psychiatrists like Gratiolet, Meynert, Wernicke, Westphal and Ziehen. Indeed, the German school of psychiatry has always had the closest relations with neurology, both in the clinic and in the laboratory, and "Professor der Psychiatrie und Nervenkrankheiten" is the rule rather than the exception in that country even today; one wonders if the sustained leadership of the Germans in the scientific development of psychiatry may not be in part due to this close relationship. Wagner von Jauregg, the first psychiatrist to win the Nobel prize, had this broad relation to his subject, as had his talented pupil von Economo.

Psychiatry up to the time of Freud was largely engaged in the description and classification of the different forms of mental disorder, which culminated in the great kraepelinian system. Since then, a more interpretative attitude has developed, in large part owing to the influence of Freud and the formulations of the psychoanalytic school. This interpretative rather than descriptive attitude has stimulated the intensive study of the individual, and offers great promise for the future in advancing knowledge of the patient and his treatment.

In this country the psychobiologic conceptions of Adolf Meyer have also had great influence in directing investigation toward the study of the individual as a whole, a broad conception of principle and method which bids fair to have a far-reaching influence on psychiatry.

In this brief and imperfect sketch of the background of psychiatry the close association with neurology may be emphasized. Psychiatrists have made great contributions to the social aspects of their field, in improvements in the care and treatment of the insane, in mental hygiene and in what is today termed social psychiatry, but the great figures in psychiatry have been distinguished by a close association with the organic neurologic aspects of their subject.

Another point that stands out in the history of psychiatry is that most of the great advances have been made in the sphere of the major psychoses. This was natural, considering the limited sphere of activity in the closed institutions, for in those days, as today, the psychoneuroses and minor psychoses constituted only a small percentage of cases in institutional practice.

It is only fair to state that neurologists in their rôle as extramural psychiatrists played no small part in contributing to these advances. They wrote treatises on mental disorders and made many contributions to the subject both clinical and pathologic, particularly in the field of the minor psychoses and the functional nervous diseases.

Thus, from the beginning, neurology and psychiatry have been closely related fields in origin, tradition and subsequent development, and it is along these lines, with modifications to meet changes in the economic and social conditions, that further progress will be made.

THE RELATIONS OF NEUROLOGY AND PSYCHIATRY

In the development of this vast field of investigation neurologists and psychiatrists have worked together, although from somewhat different avenues of approach. The psychiatrist has been concerned chiefly with the major psychoses, and the neurologist with diseases of the nervous system, each, however, with many overlapping problems of mutual interest, both psychic and somatic. Neurologists have always had important relations to psychiatry both in the hospital and in private practice, and the more profound psychiatrists have usually been trained and deeply interested in neurology. In actual practice the two fields cannot be separated—approximately 50 per cent of patients in neurologic clinics present symptoms referable to the psychic level, and in psychiatric practice nearly as large a percentage have symptoms related to organic changes in the central nervous system.

It is therefore impossible to draw a sharp line between the psychologic and the other levels of the nervous system in terms of disease; both disciplines may be said to meet on common ground, their differences of approach having been largely determined by the social factors surrounding hospitalization. This difference in the intramural and extramural life and training of the two disciplines is perhaps the largest factor in the misunderstandings and differences in point of view which exist today.

Recent years have been marked by many changes. There has been a great development of interest in psychiatry in general hospitals and medical schools, in courts, schools and colleges and in general practice. Many psychiatrists have emerged from the institutional to the extramural sphere. This has been a healthy and vigorous development and offers great promise for the future of psychiatry.

What is to be the place of neurology in this new alinement, and what are to be the relations of neurology and psychiatry in the future? What are the proper limitations of these respective fields in relation to the medical school, the hospital and in practice? These are some of the questions which are agitating leaders in both fields.

Several special articles have already appeared in the ARCHIVES dealing with these questions from the standpoint of both psychiatrist and neurologist.

It therefore seems proper to present the view held by many that the two fields are so closely allied by a common origin and purpose as to make a complete separation into organic neurology and psychiatry detrimental to the interests of both, and above all, to the subject as a whole.

In an earlier day, in this country, the term alienist was in common use to designate the specialist in mental disease, but by common consent this term has been abandoned in favor of psychiatrist. The term neurologist used to designate the specialist in nervous disease, is still in use, but others are beginning to lay claim to this designation. In the departments of anatomy of large universities the specialist in neuro-anatomy is called a neurologist and is given the title of professor of neurology. In several of the best known medical schools the title of professor of neurology is now given to the specialist in neurosurgery. One wonders, therefore, what the term neurologist will eventually represent in universities and in the public mind if its use in departments of neuro-anatomy and neurosurgery should increase to any great extent.

Another limitation, not in the name but in the function of the neurologist, has recently been promulgated from the psychiatric side. It is seriously proposed by some leaders of the new psychiatry in this country to restrict the activities of the neurologist to the organic diseases of the nervous system, to the complete exclusion of the psychic realm and even of those functional disorders of the nervous system which from time immemorial have constituted an essential part of his sphere.

In a recent contribution to this subject E. A. Strecker [1] stated:

> Somewhat arbitrarily, the psychiatric segment of the general practice of medicine may be subdivided and placed in the order of frequency about as follows:
>
> (*a*) The psychoneuroses and less well defined functional states.
>
> (*b*) Organic disease complicated by neurotic additions.
>
> (*c*) The psychopathologic implications of chronic organic disease.
>
> (*d*) The mental aspects of convalescence.
>
> (*e*) Serious psychopathologic problems in children.
>
> (*f*) Unadulterated psychoses.

1. Strecker, E. A.: Practice of Psychiatry, Arch. Neurol. & Psychiat. **31**:404 (Feb.) 1934.

A little later he asked:

> Do the psychoneuroses fall within the province of the psychiatrist or of the organic neurologist? I believe they are unquestionably within the province of the psychiatrist. Functioning as a practitioner, the neurologist dare not depart far from the structural facts of the nervous system. Naturally, an attitude of mind is induced that cannot deal effectively with the structurally unchecked data of psychopathology and psychotherapy. Presumably, broad neurologic research may eventually offer valuable aid to psychiatry, but the therapeutic exigency that exists and the results of experience both demand that in the psychoneuroses the psychiatrist prefer psychopathologic premises and psychotherapy to the less readily workable and less fruitful therapy produced by organic neurology.

One might ask why the term organic neurologist is used in this connection. When did the neurologist become purely structural in his outlook or methods of practice? The field of neurology has always included the functional nervous disorders, the neuroses and psychoneuroses. One might also ask why a knowledge of the function and structure of the nervous system should so paralyze the psychotherapeutic activities of the neurologist when the same knowledge possessed by the psychiatrist is harmless, nay even beneficial.

Adolf Meyer, in outlining the "Preparation for Psychiatry,"[2] spoke in a somewhat similar vein. He said:

> Neurology may disregard, but certainly cannot do justice to, the psychobiologic combinations and their share in the life of persons; but psychobiology does not ignore the neurologic and general physiologic and physicochemical components of the "experiment of nature" with which it deals. The neurologist can as little as any other physician afford to ignore the psychobiologic or "behavior and conduct mentation" data of the person on whom he works. Yet, making the brain and nervous system his central issue, he treats the personality functions as a secondary concern; similarly, the psychobiologist dealing with the individual as his unit treats neurology and general and special physiology and the physiochemical data as incidental or inherent data, as part of the internal and external situation or state of affairs. The "psychopathologist" may then focus on the specific factual data of mentally integrated attitudes, reactions and actions; the "psychiatrist," however, must also be able to assume general medical responsibility.
>
> To repeat, the psychiatrist must be primarily a physician, but one specifically trained to occupy himself not merely with the function of parts but with the personality function, the characteristic of which is that it is "a story in the making," a part of biographic development (in contrast to the more mechanized function of parts), operating with the help of mentation or sign function or symbolization, overt or implicit, and with its characteristic selective and constructive plasticity.

Here again it would appear that the psychiatrist, by virtue of his psychobiologic insight, is able to master the facts of neurology and to utilize all their advantages, while the neurologist is prevented by some strange structural barrier from penetrating and utilizing the mysteries

2. Meyer, A.: Arch. Neurol. & Psychiat. **30**:1113 (Nov.) 1933.

of personality and the "individual as a whole," or from using as a part of his discipline the study of the total functions of that great system which he regards as his special field.

At the last meeting of the American Psychiatric Association, the president, Dr. James V. May, took as the topic of his address, "The Establishment of Psychiatric Standards by the Association."[3] A large part of the address was devoted to the subject of the invasion of the psychiatric field by outsiders, and first among these invaders were listed neurologists. The following quotations indicate his view of the function of the neurologist:

> When this country was called upon to play a part in the great World War, the United States Army, for the first time in its history, officially recognized the importance of psychiatry. The newly created department for this purpose was put in the charge of a neurologist, and this was largely responsible for the new and important concept, "neuropsychiatry." It was soon discovered that the organic neurological conditions were not a factor of any consequence in this field, and it was placed in the hands of psychiatrists, where it very properly belongs. An analysis of the reports of the Surgeon General's Department during the period of the war showed that organic diseases of the nervous system were very infrequent. The psychoneuroses, which were, of course, of tremendous importance, belong entirely in the domain of psychiatry.

Here, once more, is the bland assumption that the neurologist is trained only in organic problems and takes no cognizance of his wider relations with the field of psychiatry and the psychoneuroses. The ungenerous statement as to the relationship of neurology and psychiatry in the World War is neither just nor true, and will be resented by psychiatrists and neurologists alike who served in the neuropsychiatric division. As a matter of fact a neurologist, the late Dr. Pearce Bailey, was the head of this division and remained the head of the department during the war. The late Dr. Thomas L. Salmon, a noted psychiatrist, was the head of the neuropsychiatric division in France, and when he returned to this country in March, 1919, he was succeeded by Dr. Edwin G. Zabriskie, a neurologist. The special hospital for psychoneuroses and the cases of shell shock was placed in charge of Dr. Sidney I. Schwab, a neurologist, whose work in this field is too well known to require comment. All of these leaders won the complete respect and cooperation of both the psychiatrists and the neurologists who served under them, and no better example could be found of the useful cooperation of these two fields of medicine than was manifested during the World War.

A little later in the same address, in stressing the subject, the following statement appears:

> Until very recently, at least, the neurological out-patient clinics, services, and hospitals have been very largely concerned in the care and treatment of the psy-

3. May, J. V.: Am. J. Psychiat. **13**:3 (July) 1933

choses and psychoneurotic disorders not belonging in the field of organic neurology. Of the 4,398 admissions to the Neurological Institute of New York during the eighteen months period ending June 30, 1932, as shown by their own official records, 54.45 per cent represented psychoses, mental deficiency, psychoneuroses, developmental defects, degenerations and scleroses involving the brain, traumatic conditions of the central nervous system, intoxications, infections, neoplasms, and various vascular disorders which almost inevitably were associated with well-defined mental symptoms. This number includes 505 cases which were for some reason undiagnosed for the time being. If we eliminate these, we are warranted, I think, in making the statement that 61.49 per cent of the total number reported by the Institute come very clearly within the classification of the American Psychiatric Association.

One might again ask when has neurology in its long history ever been limited in theory or practice to the purely organic diseases of the nervous system, and what justification is there for such an assumption on the part of the representatives of psychiatry?

It is true that some neurologists have shown little interest in the problems of psychologic medicine and have been content with the solid facts of organic neurology, but similar restrictions of interest are to be found in other fields of medicine, including psychiatry. These are, however, the exceptions rather than the rule, and the vast majority have been well trained in the mental aspects of medicine as it is encountered in hospitals and in private practice.

The names of many distinguished neurologists come to mind in this connection, such as Weir Mitchell, Mills, Dana, Putnam, Dercum, Sachs and a host of others. Psychiatrists may well remember that it was Weir Mitchell, a leader of neurology in his day, who in his epochal address to the Fiftieth Annual Meeting of the Medicopsychological Association indicated clearly the faulty psychiatric practices existing at that time in state hospitals for the insane and outlined a plan for an ideal hospital for mental diseases.

Neurologists who have considered the relations of neurology to psychiatry present another point of view. Jean Lhermitte, in his discussion of "The Training of the Neurologist,"[4] said under the caption of "Psychiatry":

> Armed with a neurologic, clinical and anatomic technic, the young neurologist must extend his knowledge into psychiatry, and for that purpose should serve for at least six months in a specialized service where he may become familiar with the psychoses, as well as with the psychoneuroses which he will have frequent occasion to treat in practice.
>
> In recent times there has been a tendency, not yet extinct, to consider the neurologist more as a physician for organic nervous diseases than as an expert capable of following and adjusting himself to the deviations of the psychoneuroses and their symptomatology, which are all the more disconcerting because they lack

4. Lhermitte, J.: Arch. Neurol. & Psychiat. **30**:405 (Aug.) 1933.

an anatomic basis. This idea, to my mind, is altogether faulty and dangerous. It is faulty because too many examples show its inaneness; and it is dangerous because, if admitted, it delivers psychoneurotic persons to physicians who lack sufficient neurologic knowledge and are incapable of discovering minimal symptoms indicative of organic changes, or worse still, it delivers them to so-called psychotherapeutists who lack medical training and give full rein to their imagination and even to their instincts.

Dr. F. M. R. Walshe,[5] in his discussion of the same subject, made the following comment, under the heading "Psychoneuroses":

. . . a primary function of the neurologist is to recognize a psychoneurosis when he meets it, and to be able to differentiate it from the syndromes of structural disease. Unless he can do this, the profoundest erudition and the most enlightened views on the genesis and treatment of the psychoneuroses are but a barren academic attainment.

It would be unnecessary to make this trite observation were it not that many enthusiastic psychotherapists ignore the truth that underlies it. Yet clearly, the discipline of a sound clinical training is as essential to the rational practice of psychotherapy as to that of neurosurgery. In this respect, the two are in one category. On the other hand, it is not to be supposed that in acquiring the skill to recognize the psychoneuroses, the neurologist has done all they demand of him. They provide one of the most exacting aspects of neurologic practice, calling for every resource of psychologic knowledge, for common sense and insight, and for clinical acumen. In the past neurologists have been too cavalier in their treatment of this problem, and it is perhaps the function of the trained neurologist in the future to import reason and the scientific outlook into a field that has for too long been the happy hunting ground of the half-trained enthusiast.

These quotations express, in a fairly general way, some of the conflicting views of neurologists and psychiatrists at the present time. The position taken by neurologists is merely a restatement of the traditional activities and clinical relations of their field since its origin. On the other hand the stand taken by psychiatrists is new, and without the support of either tradition or practice.

Certainly a glance at any textbook on mental diseases will leave no doubt as to the importance of organic disease of the brain in the genesis of these disorders. Are psychic manifestations alone the study of the psychiatrist, and are all organic factors to be left to the neurologist? And, by the same reasoning, is the neurologist to be confined to the organic aspects of the nervous system or is he to be allowed the same privilege as the psychiatrist, to study the functions, both neural and psychic, related to his field? Can the two disciplines be separated in such a crude and artificial manner, as functional (psychic) and organic, and if such a separation is made according to the principles enunciated

5. Walshe, F. M. R.: "Training of the Neurologist," Arch. Neurol. & Psychiat. **29**:368 (Feb.) 1933.

by some psychiatrists, to be consistent would they not have to abjure the organic aspects and confine themselves alone to the psychic?

History has shown that the pursuit of mental processes alone is an elusive and unstable realm, and that a sound scientific foundation is requisite. For this reason neuropsychiatry is the proper foundation for both neurologic and psychiatric medicine. The student who stands on such a broad foundation and who wishes to engage in practice or research in either field is not so likely to drift into false paths or ride each passing wave of psychologic speculation. For in the psychic field cranks, like the poor, are always with us, and sanity and caution are prime requisites.

In medicine today there is a decided trend to the biologic point of view. Man is viewed not only as a collection of organs and systems but as an individual. This movement is a general one and is not confined to psychiatry. Neurology is as much concerned with problems of constitution and all the panels that go to make up the personality as any other field. The psychobiologic and neurobiologic is the joint field of the neuropsychiatrist.

THE TRAINING OF THE NEUROPSYCHIATRIST

The training of the neuropsychiatrist should include both neurology and psychiatry. He should approach his subject from the broad standpoint of the structure, function and disorders of the whole nervous system, both in its partial and in its total reactions. This requires a thorough preliminary medical training and a sound foundation in the anatomy and physiology of the nervous system and its disorders, including the psychologic level.

In order to achieve this preparation the aspirant should spend at least one year in an approved general hospital, one year in an approved neurologic hospital and one year in an approved psychiatric hospital. He should have six months' full time work in laboratories of neuroanatomy, neuropathology and neurophysiology. His subsequent training, before he is ready for certification as a specialist in neuropsychiatry, should include five years of additional part time work in accredited neurologic and psychiatric hospitals or clinics and in private practice limited to neuropsychiatry. At the expiration of this training he should be qualified to pass an examination in neurology and psychiatry and be certified as a neuropsychiatrist.

A man so trained may then begin a more specialized career. This may lean more in the direction of the purely neurologic or the purely psychiatric, as his tastes and talents may direct, or he may devote his major interest to psychotherapy. No matter what his choice may be he will bring to his future activities the knowledge which comes of a broad foundation and a general experience with the problems of the

nervous system. And above all he will be adequately trained for the extramural practice of neuropsychiatry in its broad relations to medicine and the community.

Such a training would insure for the future neuropsychiatrist a knowledge of modern psychiatry and a broad training in the problems of neurology. It would prevent him from becoming too organically minded, and save him from a too early and exclusive preoccupation with that most difficult and complex of all fields—psychologic medicine.

The various social activities of psychiatry, as found in the clinic for mental hygiene, the court clinic, the school clinic, the clinic for child guidance and the clinic for industrial psychiatry, while serving a very useful purpose, do not in themselves give a sufficient breadth of training for the practice of extramural psychiatry, which demands a much closer association with the problems of neurology and internal medicine.

One of the faults of specialization in general has been a too early specialization in the chosen field, with an insufficient foundation in general medicine and surgery. A further danger of today in neurology and psychiatry is a too early specialization within a specialty. Men without general training enter the field of psychiatry or neurology or one of its special branches and are a credit neither to themselves nor to the field which they represent.

Psychiatry should be the queen of all the special fields of medicine. Its complexity, its capital importance, dealing with the master function of man, its manifold relations to medicine in all its branches and the very elusiveness of all mental phenomena demand the broadest and deepest training in neuropsychiatry. The psychiatrist can no more afford to ignore neurology than can the neurologist eliminate psychiatry.

Who can fathom the possibilities at the physicochemical level and their future influence on the development of psychiatry? Who can foretell the developments at the sensorimotor levels which may some day throw important light on the problems of the organic psychoses? Psychiatry cannot afford to ignore the importance of these relationships. For while the psychologic, both subjective and objective, may be the prime interest of the psychiatrist, his field is distinguished from that of the psychologist by its broad relation to disease in all its forms and to the whole field of medicine.

It is doubtful whether the categorical separation of the two disciplines which has been suggested will ever take place. It is both unnatural and illogical, and even if psychiatrists contemplate any such crude division, as would appear from some of their recent statements to be the case, neurologists do not share this point of view. They will continue to fulfil their task in the future as in the past, free to investigate and treat the disorders of the nervous system both in its functional and in its organic aspects, with the firm conviction that this association of

activities is a sound and healthy one. This does not mean that the neuropsychiatrist will not be as responsive to the life goals of the individual and to the varied sociologic relationship of this field as is the psychiatrist, but his training will serve to neutralize any tendency toward a too great absorption in these activities. A social psychiatry unrestrained may prove to be one of the future dangers of scientific psychiatry.

CERTIFICATION AS SPECIALIST IN NEUROLOGY AND PSYCHIATRY

At the present time there is a nation-wide movement for the certification of specialists in the various fields of medicine and surgery, and this may have an important bearing on the future development of neuropsychiatry.

That neurologists fully realize the importance of their relation to the psychiatric field is shown in the constitution and by-laws of the American Neurological Association, recently published in the ARCHIVES.[6] Under Article IV of the By-Laws, the preliminary requirements of candidates are as follows:

1. The candidate shall have had an internship of at least twelve months in a general hospital approved for internship by the American Medical Association, or its equivalent.

2. He shall have had an internship of at least one year in Neurology in a special hospital for nervous diseases or in a special neurological service of a general hospital recognized by the American Medical Association for residence in the specialties.

3. He shall have had one year in Psychiatry in residence in a recognized hospital for mental diseases, or its equivalent in the opinion of the Council.

4. He shall have had a minimum of six months of full time or one year of half time study of neuro-anatomy, neuropathology and neurophysiology.

5. He shall have had five years of active, official service in an accredited neurological or psychiatric hospital or outpatient department.

It is to be hoped that a similar high standard of neuropsychiatric training will be considered by the American Board of Neurology when the program is finally offered for the certification of specialists in neurology.

The American Board of Psychiatry has already under consideration certain requirements for certification in the specialty of psychiatry. The requirements for those who have practiced psychiatry less than five years are as follows:

1. A degree from a class A medical school and also a year of clinical hospital experience in a general hospital approved by the American Medical Association.

2. Evidence of not less than one year of training in a psychiatric hospital approved by the Board; or its equivalent.

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6. Riley, H. A.: Constitution and By-Laws of the American Neurological Association, Arch. Neurol. & Psychiat. **30**:1126 (Nov.) 1933.

3. Further evidence of not less than three years' additional training in psychiatry as resident physician in a psychiatric hospital approved by the Board, or full time attendance at an approved, preferably a teaching, psychiatric outpatient clinic for adults or children, or both, or in private practice confined to psychiatry.

The three year period may be divided among the several categories of training and experience indicated (except that a period of training and experience of less than six months in any one category will not be credited). Evidence of part time attendance in an outpatient psychiatric clinic may be submitted for consideration and may be credited at the discretion of the Board.

4. Report of at least ten varied cases that have been observed and treated by the applicant in the clinic or hospital with which he is connected, or in his own private practice.

5. A written and practical examination by the Board.

The candidate shall be eligible for this examination only when he has complied with the requirements above stated for Class III and has devoted a total period of at least four years to special training for, and practice of, psychiatry.

As psychiatrists now claim the right to treat as part of their discipline the large group of patients which falls in the classification of the neuroses and psychoneuroses, one finds much to criticize in this program. The neurologic hospital and clinic, which include in their varied material 50 per cent of psychiatric and neuropsychiatric cases, are not mentioned in the requirements. How is the future psychiatrist to perfect his training for practice in the field of neuroses and psychoneuroses in an institution for the insane except in a few instances in great centers in which special facilities are offered?

It is questionable whether three years of residence in an institution for committed patients would yield satisfactory training to recognize and to give intelligent treatment in this large group of cases in all the ramifications, because it is only in small numbers that intractable cases of this sort are to be found in closed institutions. Such cases are to be found chiefly in general hospitals, neurologic hospitals and clinics.

It is also doubtful whether three years of full time service in a psychiatric clinic for children would furnish the kind and variety of material which would fit a man for the general practice of psychiatry as laid down by Dr. Strecker, and this would be equally true of school, court and industrial psychiatric clinics.

One may feel certain also that three years devoted to the private practice of psychiatry alone is entirely insufficient to enter that great field envisaged by Adolf Meyer. Such a requirement would open the door to one of the greatest dangers to modern psychiatry today, the poorly trained psychotherapeutist who ekes out a career on a flimsy structure of one or the other of what Walshe has aptly termed "the seven and twenty warring sects of psychotherapy."

When one considers that psychiatry has extended its field over the whole of medicine and neurology, it cannot be denied that the list of

requirements cited is quite unsatisfactory. It should be modified so that neurologists who wish to qualify as psychiatrists may do so and still retain their status as extramural psychiatrists. The clause requiring three years of residence and the requirements of three years of full time service would eliminate most of this important group, and would tend to limit psychiatry to the intramural and full time type of man.

If such modifications are not made, then the neurologic group should require certification in neuropsychiatry and within a program which would be compatible with adequate training in both disciplines. It would, however, be more desirable to have a joint board of neurology and psychiatry and one diploma to cover both; if one were to adopt certain modifications of the programs of psychiatry and neurology, a sound system of certification could be prepared which would insure a thorough preparation in both fields. Such a plan has much in its favor and would strengthen the relations of both fields. This group of men would be truly neuropsychiatrists in foundation and training, from which firm ground further specialization could safely develop. A man so trained would be much safer in the field of practice than when proficient in only the one discipline or in a small part of one discipline.

In conclusion, I quote from Adolf Meyer's presidential address [7] to the American Neurological Association, on which occasion he advocated a change of name to the American Neuropsychiatric Association, as an expression of the mutual interests and interrelation of the two great fields. In the course of his address he said:

> As neurologists let us profess frankly that we are really neuropsychiatrists, that is, physicians with a comprehensive scope of interests and methods; and let us also see to it that the spreading of frank and intelligible views of the nature of the life problems and the psychobiologic symbolizing level becomes a necessary and obligatory concern of the rank and file of physicians, appreciated in its right importance by both physician and patient.
>
> This would not mean any submerging of either neurology or psychopathology. There will but rarely be physicians who can cover the whole field, and each investigator will have his own choice of problems. But one thing is certain: We do demand of every one a reasonable training in the entire domain, including the functions of the organism constituting the personality. We want neuropsychiatrists—not merely neurologists and not merely psychologists, but primarily physicians able to study the entire organism and its functions and behavior and more especially the share of the nervous system and of the general problems of adaptation.
>
> Let us see that the policies can be shaped by those who are able to study, who know best and work best, rather than by the sensational magazine literature and the exploiters of dissension in the neuropsychiatric camps. To attain this we have to be creative and constructive and in the front line, and we cannot trust the old policy of mere following and drifting when we come to the psychobiologic problems. We must travel under one flag and with a clear aim.

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7. Meyer, A.: Interrelations of the Domain of Neuropsychiatry, Tr. Am. Neurol. A., 1922, p. 1.

CONCLUSION

It is not easy to read the future, and he is a bold man who would attempt to foretell the relations of the workers in the special fields that have grown out of the great domain of the nervous system. That they are all working in a common cause and toward a common end is clear. It is also clear that here nature herself knows no boundaries in her infinite series of integrations. They all work as one in the interests of the organism and its environment. Men should be equally wise, and by cooperation and union unite in the common purpose.

Neurology and psychiatry appeared on the scene of medical history a little over a century ago. Neurology was the first to come of age, and now psychiatry has reached her full majority, and like a lusty youth craves greater independence and freedom from restraint.

Neurologic surgery arrived much later on the scene—a precocious child, the gift of surgery—and although not yet of age, would even now play the rôle of mother neurology herself. As neurosurgery is, however, a transplant from the great surgical field, some day father surgery will claim her as one of his fairest daughters, which will then leave neurology and psychiatry still joined to the parent stem of medicine, for better or for worse.

We should recognize this union, and while we may have our family quarrels from time to time, let us give the sons of both disciplines the family training in each. Then, when they have learned what the parents know, let them pursue their own fortunes on the tree of knowledge. Some will move in the direction of neuromedicine and some toward neurosurgery, while others will tend toward the larger and more important field of neuropsychiatry.

Abstracts from Current Literature

COMPARATIVE OPHTHALMOLOGY OF VERTEBRATES. A. ROCHON-DUVIGNEAUD, Ann. d'ocul. **170**:1 (Jan.) 1933.

In many species of selachians the visual function is limited to the perception of light and motion. Scyllaeae observed in the aquarium did not seem to see the food that was thrown to them. It was only by means of contact with the lip that they were able to notice the particles of food which had fallen to the bottom of the basin.

The retina of the teleosts is more highly developed than that of the selachians. It is more complex, is formed of finer elements, and in most species has both rods and cones; the latter are very important. Moreover, it has the richest pigmentation possible. The pigmented epithelium of the retina sheathes the external segment of the visual cells with its long pigmentary fringes, especially in the teleosts. The pigmented epithelium also shows marked movements of retraction in the dark and of expansion in the light. One wonders if the richness of the pigmented coat, protector of the visual cells, counteracts the pupillary immobility found in a great number of teleosts. The retina of the teleosts is often richer in cones than that of many mammals; in some groups there is a fovea which, however, is never so perfect as that of saurians or of birds. The fact that the predatory species see their prey from a distance and that they have a fairly well developed fovea should not lead to false conclusions concerning the visual acuity of fish. Young trout have been seen rushing on small moving larvae from a distance of from 8 to 10 cm. Even though trout prefer larvae, they sometimes eat detritus, which they vomit immediately. The sense of taste seems to correct the errors caused by imperfect vision.

Amphibia feed on larvae and little insects among which they live, without active chase, and which they recognize by their movements. The frog catches these insects by the length of its leap and the toad by the length of its tongue. Sluggish animals with immovable eyes and head immovably attached to the trunk require a large visual field but only rudimentary visual acuity. Their vision functions well in dim light, since the retina is richer in long rods and visual purple than it is in cones. In dim light the pupil, which dilates slowly but well, permits the entrance of a large amount of the feeble light found in low and dark places; but in the sunlight the pupil protects the retina by forced contraction.

Crocodiles have batrachoid customs and eyes. They are nocturnal flesh-eaters, are constantly watching for prey, on which they creep slowly after having noticed them from a short distance. In the species studied, the cones of the retina were absent and visual acuity was probably low. However, it has been observed that crocodiles have a large visual field and a well developed adaptation of the retina, and that the pupil, which is a vertical slit, is very contractile.

Serpents, diurnal animals, notice prey at a very short distance and grasp it by darting their heads forward with open jaws. They do not require very good visual acuity. If a snake, such as Zamenis viridiflavus, escapes from a man who is at a distance of from 50 to 60 meters, this does not indicate that the snake is able to distinguish him clearly at that distance. The perception of a moving mass is sufficient to frighten it.

Most ophidia, diurnal animals, are attracted by strong light, and perhaps even more by great heat. The vipers, which are seen late in the evening, are perhaps nocturnal reptiles. Their retinas have bacilliform elements beside the cones, and their pupils, which are fissure-shaped, are very contractile.

Rollinat observed tortoises on an island in a pond. The tortoises would lift their heads at a distance of 100 meters and would move away at 50 meters.

All lizards can see prey which is shown to them. Wall lizards jump at insects which are 15 or 20 cm. away, and catch them while flying. Young wall, green or stump lizards see grubs which fall onto sand and go for them immediately.

In the saurians, the structure and function of the retina are more perfectly developed. However, the visual range of this type of retina is short. The saurians do not seem to need distant vision. One is not sure, however, of the refraction of these small eyes which are so perfect in appearance. Far-sightedness appears in birds. The vulture sees a camel lying in the desert long before a man notices a vulture in the sky, although the visual conditions are to the man's advantage. The vulture's eye is not the most developed of the eyes of carnivorous birds. In fact, it is a little smaller than the bald eagle's eye, which has an anteroposterior diameter of 30 mm., even though the weight of the eagle is not half that of the vulture, and its spread is no more than 2 as compared with 2.5 meters.

As an optic apparatus, the eye of the mammals, even that of the best (the primates), is not as perfect as that of birds or perhaps of certain lizards. It has no bony scleral ring on one side of which to insert a small cornea with powerful refraction, and on the other, a large posterior segment with a wide retina. Even in the marmots and the primates, which have the best eyes of all mammals, although they are different, the degree of the corneal curve does not equal for an eye of the same dimensions that of the cornea of a bird's eye. This is due to the fact that the anterior border of the fibrous sclera of mammals, even if reinforced by circular bands, does not equal the bony ring of birds and saurians. The sclera does not supply the cornea with a similar inextensible frame which enables the cornea to keep its own curvature and to escape the general sphericity. Without this rigid ring, the intra-ocular pressure tends to impose on the entire eyeball.

However, the eye of primates has been greatly perfected. In man it is the most important sensory organ. Even though it has an excellent acuity, it is not equal to that of the bird, but it has certain qualities which the latter lacks. The bird's eye is not as mobile; sometimes it is even entirely fixed in the cranium (as in large and nocturnal rapacious animals) and has limited or no convergence. Binocular association of the central fovea does not exist in birds, owing to limitation of movement. Central binocular vision, which is necessary to the predatory species for an exact measurement of distance, has been acquired only by the development of eccentric lateral foveae in such a way that the right and the left side simultaneously receive the image from the same point. This static binocular vision, which seems to function only at a certain distance (a few decimeters), is certainly less perfect and much less flexible than the dynamic binocular vision of the primates. The latter adjusts to distance as required, even that of a few centimeters from the eyes.

From the standpoint of visual acuity a bird's eye is better than that of a primate, but the eyes of the latter are by far more widely associated for binocular vision (near or far and in all directions). The development is more evident in the nerve centers and in the mechanical structure affecting the eyeball than in the eye itself. The perfection in development has advanced from the eye to its centers.

Since the association of the ocular movements is acquired in the lower mammals, the binocular vision of primates is powerful. The fovea is found in the early stages of the development of vertebrates. However, no mammals acquired it before the primates. As soon as the fovea developed in eyes with binocular single vision, dynamic binocular vision at all distances became possible and with that all that is derived from it in the way of manual dexterity and intellectual progress. If the bird is a wing guided by an eye, the primate is a hand guided by eyes with binocular single vision. However, the eye is not everything. Man sees with the same eyes as the large apes, but he uses his visual gifts with a different brain. The eye of the gorilla is as good as, if not better than, the human eye, but the gorilla has not half as much brain as man, and there is no measure for this intellectual difference.

BERENS, New York.

EFFECT OF STIMULATION OF POSTERIOR LONGITUDINAL FASCICULUS ON OCULAR MUSCLES. NORMAN P. SCALA and ERNST A. SPIEGEL, Arch. Ophth. **9**:939 (June) 1933.

Although the anatomy of the posterior longitudinal fasciculus has been studied in much detail, definite knowledge of its function is uncertain. It is known that one-sided injuries of the bundle cause paralysis of the gaze to that side, so it is quite likely that each posterior longitudinal bundle carries impulses for conjugate movements of both eyes to its own side. The relationship or importance of the posterior longitudinal fasciculus for the conduction of vestibular impulses to the ocular muscles is not wholly clear. One of the authors demonstrated experimentally that extensive bilateral injuries of the formatio reticularis, reaching close to the posterior longitudinal fasciculus, did not prevent the genesis of horizontal as well as of vertical nystagmus produced by labyrinthine stimulation.

One has to assume, therefore, that the posterior longitudinal bundle is not the only pathway for the labyrinthine impulses to the nuclei of the ocular muscles, but that a second pathway exists in the formatio reticularis. If one or the other is destroyed, the one remaining is probably still able to conduct vestibular impulses to the ocular muscles. Spiegel recently severed the right posterior longitudinal fasciculus at the level of the sixth nucleus and also just behind it, extirpated also the left external ocular muscles, with the exception of the internal rectus, and was then no longer able to produce a reaction of the left eye by rotation of the animal. It is therefore likely that the posterior longitudinal fasciculus connects the sixth and third nuclei of the same side, thus innervating the homolateral external rectus muscle, whereas the impulses to the opposite internal rectus reach this muscle by crossing roots of the third nerve. The work of Klossowsky and Levikowa is not wholly in accordance with the experimental findings of Spiegel. Briefly, if the experiments and the theories of these two men are correct, the posterior longitudinal fasciculus should carry in the cranial part of the pons ascending fibers to the homolateral internal rectus. Further work is undeniably necessary.

The relation of this system of conduction of impulses through the posterior longitudinal bundle to the innervation of horizontal movements of the eye is questionable, but the same relation to vertical movements of the eye is still more hypothetic. Spiller demonstrated that lesions in the neighborhood of the aqueduct or in the cranial part of the pons may produce paralysis of gaze in the vertical direction. On the other hand, Poetzl and Sittig assumed that lesions of the ventrocaudal part of Deiters' nucleus are able to produce skew deviation.

The notation of further instances is unnecessary. They all emphasize the fact that continued observations must be made to clear up the controversy and inconsistencies present relative to the conduction of impulses by the posterior longitudinal fasciculus. It was because of this that Spiegel carried out the experimental work he did. Because of its interest, the technic of the laboratory work is included in the abstract.

The experiments were performed on cats. Under ether narcosis both carotid arteries were clamped, the skull was opened on both sides, and the forebrain and interbrain were extirpated, with the exception of the most ventral parts, to avoid injury to the nerves of the ocular muscles. A lesion of the corpora quadrigemina was also carefully avoided. Then the narcosis was discontinued, the tentorium was removed, the underlying dura was opened, and the lobus anterior cerebelli was slowly separated from the colliculus inferior by small pledgets of cotton wool, and elevated from the anterior floor of the fourth ventricle. In further experiments, not only the cranial part of the fossa rhomboidalis but the whole floor of the fourth ventricle was exposed by a total extirpation of the cerebellum after removing the occipital bone with the membrana atlanto-occipitalis and also carefully separating the posterior part of the cerebellum from the fossa rhomboidalis. The diffuse electrode was fastened on the abdomen; the stimulation was made by Sherrington's single wire electrode. Only weak faradic currents were used, just sufficient to produce a slight contraction if applied to an exposed muscle. To

observe the movements of the eyes, the membrana nictitans was removed, the palpebral fissure was enlarged by an incision at the external canthus, and the eyelids were separated by eye specula.

The report on one of the laboratory animals is included in its entirety to illustrate the procedure as well as to show some of the findings on which the authors based their conclusions.

Report of Test on One Animal

	Stimulation	Effect on Left Eye	Effect on Right Eye
12:40	Right posterior longitudinal fasciculus in cranial part of pons	Downward	Upward
12:52	Left posterior longitudinal fasciculus.....	.....	To left
12:53	Right posterior longitudinal fasciculus.....	Downward after removal of the electrode	First upward, then downward (after removal of electrode)
1:00	Right posterior longitudinal fasciculus.....	Slightly upward	First upward, then downward after removal of electrode
1:02	Puncture of left posterior longitudinal fasciculus	Upward	Upward
	Second puncture of this bundle on left side in cranial part of pons	.....	To left
1:05	Right posterior longitudinal fasciculus.....	.....	Upward
	Left posterior longitudinal fasciculus behind puncture	No sure reaction (slight undulation)	
1:10	Puncture of right posterior longitudinal fasciculus	Downward	Downward
1:12	Right posterior longitudinal fasciculus behind puncture	No reaction	
1:15	Right posterior longitudinal fasciculus behind puncture	.....	Slightly upward
1:16	Transverse section through both posterior longitudinal fasciculi behind punctures	.....	Upward
1:20 1:26	(No reactions of eye to repeated stimulation behind transverse section)*		

* Respiration was still regular.

In their experimental work, the various findings were checked anatomically by subsequent histologic examinations to clear up, as much as possible, the logical objections which would certainly arise. The most important conclusions reached were that these experiments supported the assumption that the posterior longitudinal fasciculus carries ascending impulses to the crossed internal rectus muscle, and that besides movements of both eyes in the same direction, dissociated movements of the eyes are also observed, particularly horizontal movements of the opposite and vertical movements of the homolateral eye.

SPAETH, Philadelphia.

PSYCHOGENIC REACTIONS INCLUDING THE SO-CALLED COMPENSATION NEUROSES. M. REICHARDT, Arch. f. Psychiat. **98**:1 (Nov.) 1932.

In this contribution Reichardt presents his concept of the nature of "compensation neuroses" and discusses a number of opposing theories. His arguments are directed mainly against authors who consider this reaction type as a new form of neurosis which arises out of the development of certain social institutions and for which social forces are responsible (compare von Weizsäcker, V.: Ueber Rechtsneurosen, *Nervenarzt* **2**:569 [Oct.] 1929; Soziale Krankheit und soziale Gesundung, Berlin, Julius Springer, 1930). The main thesis is that compensation reactions are not true neuroses but belong to the psychogenic wish or goal reactions (*Wünsch oder Zweckreaktionen*). They belong to the hystetical reactions

in which fearful expectation of the possible occurrence of certain symptoms may serve as the causal agent (expectation neuroses). The symptoms are sustained either in order to gain compensation or to avoid a difficult situation (war). Often the accident or trauma serves as an apparent reason for a distress which may actually be due to a preexisting but not recognized physical disease or to a series of conflicts under which the patient was just about to break.

Certain personality traits, such as suggestibility, tendencies toward "überwertige Ideen," or repressions, all of which may occur in normal life, may, if exaggerated, become predisposing factors. There are certain persons in whom emotional experiences lead to pronounced changes in the vegetative system. Such reactions, however, should not be called psychogenic. Vegetative stigmatization in itself has nothing to do with psychogenic reactions. Only in cases in which the physical condition is used by the patient for avoiding a difficult situation or for gaining other advantages should the condition, according to the author, be called psychogenic. This means that depressions and other mental disturbances which can be traced back to recent precipitating experiences should not be called psychogenic.

The author believes that the variation from the normal in such reactions is not a qualitative but a quantitative one; it cannot be looked on as a disease process, and therefore the patient is not entitled to compensation. In true simulation the wilful intention to decive and gain advantage by the demonstration of physical symptoms is obvious. In the psychogenic reactions this intention is disguised, and the patient hides it from his own consciousness. The author regards as true neuroses only reactions which develop on the basis of true autosuggestion, or under the influence of "überwertige Ideen" for which the patient is not responsible. He cites several examples of what Kretschmer called "wilful reinforcement of reflexes." In his opinion a large number of the physical and mental manifestations in so-called compensation neuroses were at a voluntary level in the beginning and only gradually by practice became hidden from consciousness.

In compensation neuroses it is important to distinguish between the true results of the accident or traumatic experience and the results of the expectation to obtain compensation. The immediate results of an accident in the form of concussion form a clear clinical picture, which, according to the author, is easily recognized. Shock should be considered as real only in the case of vegetative shock, which is seen in surgical operations in the form of dangerous collapse of life activities. More prolonged neurotic manifestations, which usually are contributed to shock, have nothing to do with it. The results of the emotional upheaval accompanying severe accidents and life-threatening situations usually disappear within a quarter of a year. Only occasionally does one observe a general physical depletion with loss in weight and mild depression of mood, which also disappears within this time. The author warns against overestimating the significance of irritability, memory disturbances and other defects in cortical functions. Only too often he found that the examiner became a victim of his own suggestions to the patient. Ganser's syndrome and Raecke's pseudodementia, according to the author, should be considered as true simulation. Such patients should be able to meet court trials and should be dealt with as mentally normal persons.

Reichardt admits the necessity of treatment in many cases but believes that the physician should separate clearly his function of physician from that of judge in the dispute between the patient and the insurance agency when the only goal is to determine whether the patient's complaints are the results of an accident or not.

In method of treatment he stresses a "strong moral" position on the part of the physician. It is important to show the patient as soon as possible that his demands are not legitimate, and that the physician does not hold out any hope for compensation for the type of complaint which he presents. Psychoanalysis may be used occasionally, but it is of no material help in most psychogenic reactions. In a chapter on the psychology of physicians who have to render an opinion in court, he points out the personal element which makes the physician inclined to be humane and somewhat sentimental in his efforts to help the patient, and which has led to so much confusion of opinions in this field. He requests more careful

education of the medical student in this part of medical psychology and says emphatically that the physician who first treats the patient should make a very detailed record of the findings within the first days and weeks after the accident.

MALAMUD, Iowa City.

HEADACHE AND PAIN IN INFLAMMATION OF NASAL SINUSES. HERBERT TILLEY, Brit. M. J. **1**:549 (April) 1933.

Pain is a frequent and sometimes the chief subjective symptom of chronic inflammation of the nasal sinuses. It may be generalized or confined to more or less restricted areas. It should be emphasized that the air cells on both sides may be in a state of chronic inflammation, even of the suppurative type, and yet there may be no history of diffuse or restricted pain in the head. The nasal sinuses, or air cells, and especially the ethmoid, frontal and sphenoid cavities, lie in such close relation to each other that it is unusual to find inflammation confined to any one of them. It is important to remember that the air cells, as well as the whole face area and most of the deeper structures of the head, derive their sensory innervation from the fifth nerve.

When pain happens to be a symptom of chronic nasal sinus inflammation its cause is frequently the retention of pathologic secretions under tension. Pain or headache may be complained of, however, even when drainage is free, or following the natural opening of the sinus into the nasal cavity. This may be due to at least two causes: (1) the presence of a secondary chamber or loculus within the parent air cell and not freely discharging into it; (2) a hypersensitive condition of the inflamed mucous membrane of the sinus in a patient whose deep trigeminal nuclei have a low threshold of resistance to afferent impulses. The explanation of the phenomenon in the latter group may be that it results from a mild toxemia arising from the infected sinus or from some independent constitutional factor. In other instances headache or pain is produced by mere pressure on sensory nerves in the vicinity of the lesion.

Referred pains are common. It is Tilley's experience that pain is more likely to be of the localized or of the referred variety when only one sinus is affected, whereas a general headache is more common when more than one air cell is inflamed. In chronic antral inflammation, whether of dental or of intranasal origin, the pain will often be experienced in the cheek area or referred to a region corresponding to the supra-orbital foramen and the distribution of the supra-orbital nerve. In others, the pain may affect the whole of the corresponding side of the head. In chronic inflammation of the ethmoid air cells, pain is most often felt in the region bordering on the inner side of the orbit and at the root of the nose. From a diagnostic point of view, such pain can often be relieved immediately by the application of cocaine to the internal and external branches of the nasociliary nerves in the upper anterior regions of the nasal fossae. In other instances the pain may be felt over the vertex, or there may be a feeling of pressure in that locality.

The pain of frontal sinusitis is usually localized in and immediately around the region of the sinus and is definitely aggravated if pressure is applied upwardly and inwardly on the inner region of the floor of the air cell. In regard to this sinus, a "vacuum headache" may occur. It is due to the narrowing of the fronto-nasal duct leading from the sinus into the nasal cavity, brought about by the encroachment of the adjacent ethmoid cells through which the duct passes. Absorption of the air in the sinus brings about a vacuum and this induces a dull frontal headache. This is much increased by the sustained use of the eyes.

In involvement of the sphenoid sinus the most frequent location of pain is over the occiput; next in order is the vertex, and, occasionally, the region supplied by the supra-orbital nerve. If the vidian nerve becomes involved by inflammation of the outer wall of the sinus and neuritic changes reach Meckel's ganglion, the sphenopalatine ganglion syndrome may result. There follow neuralgic pains in the supra-orbital ocular, superior maxillary, mastoid and occipital regions, and

in some cases they may extend to the shoulder blade, the arm, and even to the tips of the fingers. These symptoms may be accompanied by lacrimation and sneezing attacks. When the diagnosis has been established, injections into the ganglion will sometimes cure the pain. Another symptom of sphenoid sinus disease is pain over the corresponding mastoid region.

In his summary and conclusion Tilley emphasizes the importance of correct diagnosis in these conditions, especially when physical intervention is contemplated and required. The necessity for a complete examination is illustrated by brief reference to a patient who was considered for an operation because of chronic antral suppuration but who died on the following day in diabetic coma.

FERGUSON, Niagara Falls, N. Y.

CEPHALIC ZONAS. J. REBATTU, P. MOUNIER-KUHN, J. DECHAUME, P. BONNET and A. COLRAT, Rev. d'oto-neuro-opht. **11**:333 (May) 1933.

Laryngeal paralysis in zona is always accompanied by otic zona and facial paralysis. In Federici's case the evolution of the paralysis was followed from its beginning, and it strikingly confirmed the law of Semon-Rosenbach. Rapid recovery is the rule. Knowledge of lingual paralysis is limited to the observation of Faure-Beaulieu, Mathieu and Cord in 1931: In the course of a zona of the face, neck, ear and tongue, homolateral paralysis of the tongue, followed by hemiatrophy with partial reaction of degeneration of the hypoglossal nerve, was seen. Paralysis of the masticators would be a logical expectation, but no instance of it was found. Paralysis of the muscles of the neck is very rare.

Sensorial disturbances of the eighth pair in zona of the geniculate ganglion may involve both the cochlear and vestibular divisions or either separately. In eighty-two observations of involvement of the eighth nerve, cochleovestibular disturbances represented 44 per cent, cochlear disturbances, 45 per cent, and vestibular derangements, 11 per cent. The order of the appearance of the symptoms is: neuralgic pains, eruption, facial paralysis and equilibrial and auditory troubles. Cochlear symptoms may be represented by auditory perversions, hyperacusis or hypo-acusis or diplacusis. Systematic examination is often necessary to discover them and they are of the nerve type. Auditory disturbances disappear concomitantly with regression of the facial paralysis. The characteristic sign of vestibular disturbance is vertigo, which is often accompanied by nausea and vomiting. The attacks recur for months, and are accompanied by loss of equilibrium and nystagmus directed toward the healthy side. Vestibular examination reveals either a hyperexcitability or hypo-excitability of the labyrinth. Recovery within two months is the rule, although exceptions occur. Recrudescence may occur after apparent cure. Probably the primary infection of the geniculate ganglion is propagated along the sheaths to the ganglia of Scarpa and of Corti. When suppurative otitis media occurs as a complication, it is explained by rupture of a vesicle, leading to perforation of the membrane.

Theoretically, disturbances of taste should occur in zona affecting the fifth, seventh and ninth nerves, but in practice they are encountered only in zosterian facial paralysis.

No indubitable facts have been adduced to prove involvement of the olfactory nerves in zona. Disturbances of olfaction may be due to the mechanical obstruction offered by swelling of the mucosa of the turbinates and septum. Disturbances of the sympathetic system occur, the syndrome of C. Bernard and Horner being an example. Any part of the system may be involved from the cutaneous terminations to the bulbar segments, which explains the different manifestations from one case to another.

Alternate hemiplegia, with or without paralysis of the third nerve, has been observed and is due to lesions of the central nuclei. It occurs only in the aged. Schiff and Russell reported a case of zonal encephalitis and André Thomas and Buvat reported one, and cases of zosterian cachexia, all fatal, have been noted.

The pathogenesis of these conditions is not clear; they may be due to circulatory accidents occurring in the aged with fragile vessels following vasomotor disturbances produced by zona.

Clinical forms of cephalic zonas are classified as cervical, facial, buccopharyngeal, ophthalmic and auricular. Most often zonas of the face are limited to the region of the superior or the inferior maxillary nerves. A diagnosis of buccopharyngeal zona is often possible only when there is association of paralytic phenomena or cutaneous eruption. Auricular zonas are divided into simple, auricular zona with facial paralysis, auricular zona with auditory disturbances, and associated auricular zonas (the most common association being with one or more branches of the trigeminus).

Treatment is confined practically to relieving the symptoms and treating complications, although serum from convalescents, antidiphtheritic serum, autohemotherapy and arsenic have been used with good effect in shortening the attack.

DENNIS, Colorado Springs, Colo.

REVIEW OF THE WORK OF THE PSYCHIATRIC INSTITUTE AND HOSPITAL DURING THE PAST YEAR. CLARENCE O. CHENEY, Psychiatric Quart. **7**:16 (Jan.) 1933.

In an address to the quarterly conference, Cheney reviews the recent work of the New York Psychiatric Institute. Of particular interest is the discussion of: (1) psychoses among children, (2) dementia praecox, and (3) fever therapy.

Forty-five children (under the age of 16) were admitted to the Institute during the year. Cheney believes that the majority of psychiatric problems in children below the immediate prepuberal ages are better treated at home than in an institution. Of these 45 children, 19 were classified as having a psychopathic personality or psychoneurosis; 5, juvenile dementia praecox; 7, dementia praecox; 2, manic-depressive psychosis. Of the 45 children, 2 were discharged as recovered, 15 improved and 6 unimproved—the others still being in the institution at the time of the study. The unimproved group consisted largely of patients with juvenile dementia praecox and emotional instability associated with chronic encephalitis.

Symptoms of and treatment for juvenile dementia praecox constitute the chief research problem of the pediatric division of the hospital. Contrary to many other authorities, Cheney and his associates believe that the psychosis does appear below the age of 10. The value of a carefully organized daily routine, including school, organized play, games and gymnastic activities is stressed, the procedures being designed largely to objectify interests. In most cases it is the appearance of a definite interest in some daily activity which marks the beginning of improvement in these patients. Many children with schizophrenia appear to be, and have been institutionalized as, feebleminded. The author pleads for better care in the diagnosis of the condition of, and classification of, children who appear at a casual glance to be mentally defective, believing that more thorough psychiatric study will reveal many early examples of dementia praecox in this group.

The care of adults with catatonic dementia praecox is also discussed. The Institute staff converted a dormitory into a huge oxygen chamber, and ten patients with catatonic schizophrenia were placed there. These patients lived continuously in the atmosphere of 50 per cent oxygen and 3 per cent carbon dioxide; five received, in addition, daily treatments of from 15 to 40 per cent carbon dioxide during a two minute exposure daily. A third group received the latter treatment without living in the oxygen chamber. No appreciable clinical improvement was noted, and the experiment was discontinued with the belief that neither the oxygen nor the carbon dioxide inhalation technic had proved of significant value. From a study of a large series of patients with schizophrenia, it is Cheney's belief that a fair proportion of them gain and maintain an adequate type of adjustment, quite apart from the nature or duration of the symptoms or even from the depth of

the regression. Photographs of some of the patients, taken while asleep, show that they assume normal positions and change them with normal frequency—an observation which tends to show that catatonic postural rigidities are of cerebral or psychic origin rather than toxigenic.

High frequency apparatus was used to produce fever in 98 patients with dementia paralytica. An analysis of 42 of these shows 6 deaths, 24 cases of improvement and 12 of remission. Because of the apparent increase in receptivity to arsenic shown by heated brains of mice (work of Lebedewa and Galanowa), the Institute staff intends to treat patients with dementia paralytica with high frequency and tryparsamide simultaneously.

DAVIDSON, Newark, N. J.

EXPERIENCES WITH ENCEPHALOGRAPHY WITH SPECIAL REGARD TO THE INSUFFLATION OF AIR BY CISTERNAL (SUBOCCIPITAL) PUNCTURE. ERIK ASK-MARK, Acta psychiat. et neurol. **7**:22, 1932.

Encephalography and ventriculography have been carried out on eighty-five occasions on seventy-six patients. The method employed in fifty cases was that of cisternal (suboccipital) puncture; in eighteen, that of lumbar puncture, and in fifteen, that of ventricular puncture. In twenty-seven cases epilepsy was diagnosed; in twenty-eight, tumor cerebri, and in twenty-one, other conditions such as vascular lesions and pseudotumor cerebri. Pathologic conditions detectable by encephalography were present in forty-one cases, encephalographically normal conditions in twenty-seven and questionable conditions in seventeen.

Ask-Mark concludes that the insufflation of air by cisternal puncture should be used as the normal method. It is less dangerous than ventriculography and lumbar encephalography; it is technically more simple than ventriculography and hardly more difficult than lumbar puncture; it is subjectively less disturbing than lumbar encephalography; the roentgenograms obtained are at least as clear as those obtained by the injection of air into the spinal sac and essentially as satisfactory as those obtained by ventriculography.

Ventriculography should be used only if sufficient information cannot be obtained by suboccipital encephalography. However, the latter procedure has certain complications and dangers and for this reason should be used only if the usual neurologic and roentgen examinations do not establish a conclusive diagnosis. The complications are headache, vomiting and other phenomena such as slowing and weakening of the pulse, changes in the color of the face and sweating. These seem to be due to meningeal irritation by the air and to the increase in intracranial pressure. The intensity and duration of these complicating symptoms depend on three factors: (1) the method used (signs of meningeal irritation occur earlier and perhaps are more intense with the cisternal method, but occur, although they appear later, with the intraventricular); (2) the amount of air injected (this seems to produce a slighter reaction by the cisternal than by the other two routes); (3) individual differences in susceptibility to reactions. The dangers may be due to: (1) trauma by the needle (e. g., puncture of large vessels), but this is none the less possible by the intraventricular route than by the cisternal, particularly if in the latter the needles are kept in the midline; (2) infection; (3) sudden changes in pressure (depending somewhat on the character of the case, cerebral tumor offering the greatest danger from this cause). Of fifteen patients in whom the intraventricular route was used, two died; of eighteen in whom the lumbar route was used, none died; and of fifty in whom the cisternal route was used, none died.

Encephalography gives valuable information in establishing the absence of pathologic processes in cases in which they are suspected and in revealing the presence of certain abnormalities, particularly distention of the ventricular system, displacement of the ventricular system or of the great fissures of the brain, defective filling of the ventricular system with air and cyst, which is demonstrated if filled with air.

PEARSON, Philadelphia.

VESTIBULAR APPARATUS IN NEUROSIS AND PSYCHOSIS. PAUL SCHILDER, J. Nerv. & Ment. Dis. **78**:1 (July) 1933.

Through the research of Flourens, Breuer, Mach and others it is known that the vestibular apparatus is responsible for the phenomena which occur after turning and for the perception of acceleration. The semicircular canals enable one to perceive circular movement and possibly also the progressive movements of the body, although the otoliths are more important in the latter function. Reflexes of posture connected with the labyrinth influence the muscle tone of the body. The vestibular sense is thus more closely related to primitive motility than is vision, but it works synesthetically with vision, audition and touch. Though attitude, posture and motion form an unconscious background of experience, they are none the less important, for they are apt to have a stronger influence on the vegetative nervous system and hence may produce psychic changes. Spitzer has differentiated two functions of the nervous system, idiotropic and oikotropic; the former coordinates single parts of the body and the latter correlates the body with the outer world. The lateral vestibular nuclei are between the oikotropic and idiotropic in their functions, while the median nuclei are only idiotropic.

Schilder concludes from previous and recent studies of patients with vestibular lesions that the vestibular apparatus has an influence on the visual field, which may be homolateral. Darkening and concentric narrowing of the field are common. There is a multiplicity of apparent movements which are only partially dependent on the nystagmic movements of the eye and may be caused by central as well as by peripheral lesions. These apparent movements have a great tendency to transformation. The perception of direction can also be changed by vestibular lesions, and frequently these transformations are from one plane to another. Polyopia and micropsia belong also to the symptomatology of vestibular lesions and are usually homolateral to the side of the lesion. Under vestibular influence part of the substance of the body may be dissociated subjectively from the rest of the body, and the subjective sense of heaviness and lengthening of the body is dependent on the vestibular apparatus. Optic and tactile images, and tactile and optic eidetic pictures can be influenced by vestibular irritation in a similar way as after-images. In hallucinations vestibular influences change the appearance and add movements to the picture. Multiplicity of hallucinations, macropsia, micropsia and dysmetamorphopsia indicate a vestibular influence on hallucinations. Vestibular changes affecting the unity of the postural model of the body are studied in connection with delirium tremens and alcoholic hallucinations.

Schilder studied a case of intoxication from barbital and one of eclamptic psychosis from this point of view. He concludes that dysfunction of the vestibular apparatus is often the expression of two conflicting psychic tendencies—hence the occurrence of vertigo in almost every neurosis. It is a danger signal that the ego cannot exercise its synthetic function in the senses, but it occurs when conflicting motor and attitudinal impulses cannot be united any longer. Hence, the vestibular apparatus may be regarded as an organ the function of which is directed against the isolation of the diverse functions of the body. HART, Greenwich, Conn.

RELATIONSHIP BETWEEN TRAUMA SUSTAINED AT BIRTH AND ENCEPHALITIS IN CHILDREN. SOL ROY ROSENTHAL, Arch. Path. **16**:33 (July) 1933.

In the experience of most pathologists there appear from time to time cases of encephalitis in children which are not associated with acute general infections or well established forms of encephalitis and for which the etiology remains obscure. In the literature the cases are classified as encephalitis of idiopathic origin, as atypical forms of encephalitis, as insidious encephalitis and as encephalitis following pyogenic infections. In many of these cases the location of the lesion, the fatty changes and the cortical atrophy usually associated with paralysis cannot be wholly explained by the infection. One must then seek for some other underlying pathologic process. This is accomplished only with great difficulty, in that the reactions of the parenchyma of the brain and the perivascular connective tissue

to inflammatory, toxic and traumatic irritants are similar and at times impossible to differentiate. Coupled with this handicap is the fact that there is no definite way of determining the age of a lesion in the brain.

There were many similarities in the three cases in one family which were reported. The cases were characterized by repeated attacks of symptoms referable to the central nervous system. The onset was insidious, with symptoms of slight headache, nervousness and vomiting, so that attention at first was not drawn to the brain. The actual attacks were more acute, with paralysis in the first case and extreme nervousness and vomiting in the first and third cases. The terminal syndrome was most severe, with semiconsciousness and convulsions.

It has been suggested that in many cases of so-called idiopathic and atypical types of encephalitis in children, the underlying cause may be the injuries sustained at birth. With the brain in a state of lowered resistance, cryptogenic or obvious infections may initiate encephalitis. The clinical history in such cases may indicate a difficult or prolonged labor, although the morbidity in so-called normal labors is sufficient. The children may have signs referable to the central nervous system from birth, or these signs may be absent or so slight as to be overlooked. Repeated attacks of encephalitis should probably be considered pathognomonic, more especially if associated with paralysis. The temperature, the findings in the spinal fluid and the number of leukocytes in the blood depend on the severity of the superimposed infection. The prognosis is not necessarily one of a fatal outcome and is dependent on the severity of the underlying injury and the virulence of the invading organism or toxin.

Microscopically, the presclerotic or the sclerotic foci, with colloid deposits or deposits of calcium and the formation of cysts, are suggestive of trauma sustained at birth. These lesions may be found in any portion of the brain, but characteristically in the frontal and occipital lobes, in the portions drained by the vena terminalis and the vena lateralis ventriculi and in the basal ganglia. The secondary process may be true encephalitis or pseudo-encephalitis.

WINKELMAN, Philadelphia.

TUMORS OF THE PITUITARY GLAND AND ITS NEIGHBORING STRUCTURES. L. M. DAVIDOFF, Psychiatric Quart. **7**:72 (Jan.) 1933.

The acromegalic symptoms of pituitary disease are due to hyperfunction of the acidophilic cells; the sexual dystrophies are probably an expression of basophilic involvement. When seen in the same patient the growth is probably acidophilic, the genital symptoms being brought about by destructive pressure on the basophilic cells resulting from expansion of the tumor. Disturbances in fat and urine metabolism are probably due to involvement of the hypothalamus.

Davidoff lists the lesions of the pituitary region in order of frequency: The commonest is the hypophyseal adenoma, next in order are craniopharyngeal pouch cysts, suprasellar meningiomas, chiasmic gliomas, and aneurysm of the circle of Willis. Rarer disorders, such as chordoma, angioma and cholesteatoma are mentioned. The symptomatology of each of the commoner lesions is outlined. Pituitary adenoma is diagnosed by optic chiasm symptoms: headache, adiposogenital involvement, growth disturbance and roentgenogram evidence of sellar erosion. Cysts of the craniopharyngeal pouch occur usually in early life and are manifested by hypothalamic, pituitary and chiasm symptoms. The frequency of shadow-casting calcium deposits in the cyst wall is a diagnostic aid. The suprasellar meningioma arises from the tuberculum sellae, giving rise to hemianopia without functional pituitary signs. Glioma of the chiasm causes early and extensive visual defect, and is indicated on roentgenograms by an anterior extension of the sellar fossa passing under the clinoid.

Chronic arachnitis is difficult to diagnose before operation. The symptoms are due to thickening of the arachnoid with pocketing of fluid in the leaves of the membrane. Aneurysms of the circle of Willis can be diagnosed by the paroxysms of unconsciousness, meningism and bloody spinal fluid.

The surgical approach to the hypophysis is discussed in some detail. The transsphenoidal route of Cushing and Frazier is serviceable only in strictly intrasellar lesions. Approach by craniotomy, lifting the frontal or temporal lobe, is becoming increasingly popular. Sometimes an adenoma may be removed, but more often only enough of it can be taken out to relieve pressure. Radiotherapy is used to destroy the remaining fragments. No other primary pituitary lesion is radiosensitive, this technic being available therefore only in the instance of adenoma. The craniopharyngeal pouch cyst and the chiasmic glioma are unfavorable lesions from the therapeutic point of view. Surgical approach is unsatisfactory, yet no other method is possible. Suprasellar meningiomas, on the other hand, can be removed by the electric loop or shelled out surgically, gratifying results often following the treatment of this lesion.

DAVIDSON, Newark, N. J.

PHYSIOLOGICAL LEUCOCYTOSIS: THE VARIATION IN THE LEUCOCYTIC COUNT DURING REST AND EXERCISE AND AFTER THE HYPODERMIC INJECTION OF EPINEPHRINE. H. E. MARTIN, J. Physiol. **75**:113 (June 21) 1932.

Convalescent patients without organic lesions were chosen by Martin as subjects. They were confined to bed, with a light diet, during the whole of the day of the experiment. Six samples of blood were taken from each patient, the period from 12 to 2 p. m. being avoided. It was found that the leukocytes are at a minimum during the period when the patient's condition approximates most nearly complete physiologic repose, and tend to rise with increase in mental and physical activity. The leukocyte count, like the temperature, tends to rise in the late afternoon and early evening, this being regarded physiologically as the most active part of the day.

The effect of exercise on the peripheral blood was investigated. The total leukocyte count exhibited a rise varying from 15 to 48 per cent of the resting value, and returned to normal after about thirty minutes of rest. Further exercise produced another rise in the count similar in type and degree to the first. The rise in the total count was due to an increase in the number of all types of cell, but the lymphocytes showed the greatest increase. In order to determine whether the leukocyte increase during exercise was due to the passage into the blood stream of immature cells from the bone marrow, Schilling counts were carried out in selected cases. The increase in the polymorphonuclear cells was made up mainly of the mature leukocytes, but there was a small increase in the "band" forms, which indicates that the cells were probably liberated from the bone marrow. Since exercise is a normal physiologic condition, a large influx of immature cells into the circulation is not to be expected, as they are rarely present in normal blood. One of the subjects in the group was a splenectomized patient, who reacted to exercise exactly as a normal subject.

As it is known that epinephrine is secreted into the blood during excitement and exercise, it was decided to investigate the effect of the subcutaneous injection of epinephrine on the leukocyte count. The hypodermic injection of epinephrine is followed by a leukocytosis in which all types of cells show an increase, but that of the lymphocytes is most marked. It is similar to the leukocytosis following exercise, suggesting that the latter is produced by autogenously secreted epinephrine. Martin concludes that these leukocytes arise from two sources: The lymphocytes are in all probability derived from the lymph glands, which contract under the influence of epinephrine, and the granular cells are washed out of the bone marrow by the increased flow of blood owing to cardiac stimulation by epinephrine.

ALPERS, Philadelphia.

DARK ADAPTATION IN THE ALBINOTIC EYE. DOROTHY J. SHAAD, Arch. Ophth. **9**:179 (Feb.) 1933.

This is an extensive article, discussing dark adaptation in the albinotic eye. The author thoroughly covers the literature and its relationship to albinos.

In the experimental work, a Nagel adaptometer was used to follow the course of adaptation for half an hour, with monocular observations after one, five, ten,

twenty and thirty minutes in the dark. Each subject was light adapted for five minutes before the test. Shaad, in her experimental work, paid no attention to the size of the pupil. (The abstracter regrets this omission, because he thinks that in a study as academic as this a factor which can be as well controlled as the size of the pupil, and hence the measurement of retinal illumination, should have been considered.)

A comparison of the albinotic and normal adaptation was done on the group, even though it was small. The ratios may be determined in terms of "chances that the obtained difference represents a true difference greater than zero."

Comparison of Albinotic and Normal Adaptation

Minutes in the Dark	Ratio	Sensitivity of Albino
1	1:64	94 chances per hundred that any albino is less sensitive than a normal person after one minute in the dark
5	1:41	92 chances per hundred that any albino is less sensitive than a normal person after five minutes in the dark
10	1:63	94 chances per hundred that any albino is more sensitive than a normal person after ten minutes in the dark
20	2:96	99.9 chances per hundred that any albino is more sensitive than a normal person after twenty minutes in the dark
30	2:64	99.6 chances per hundred that any albino is more sensitive than a normal person after thirty minutes in the dark

In the summary the author states some rather definite conclusions. They are that albinos adapt less rapidly at first, but become somewhat more sensitive than normal subjects after ten minutes in the dark, and remain somewhat more sensitive up to the end of the thirty minute test. Further, that while lack of normal pigment in the albinotic eye may be a physical factor causing the initial retardation in the albino sensitivity, still it does not prevent the progress of dark adaptation.

SPAETH, Philadelphia.

PAIN. DAVID WATERSTON, Lancet **1**:943 (May 6) 1933.

The observation that the epidermis is painless, despite the fact that it contains afferent nerves, afforded Waterston a new standpoint from which to approach the problems of production of pain. He previously had had the necessary training in the analysis of sensation; the experiments were carried out on himself. The general procedure was simple. A sharp needle was introduced through the skin and passed into underlying tissues to different depths; muscles, tendons, periosteum and veins were stimulated, as well as the wall of an artery. The more important facts which were observed give a clear conception of the pain apparatus. There is no doubt that in the skin the pain apparatus is separate from that of tactile sensation. This conclusion has a bearing on the much disputed problem of peripheral sensibility and especially on Head's theory of its mechanism. The observations in the present instance have not dealt with perceptions of temperature, but there is now no doubt of the correctness of Head's view that the tactile and the pain mechanisms of the surface of the body are separate anatomically. The deeper tissues of the body can as a rule give rise to pain, and arteries especially are a source of acute pain from mechanical stimulation. It seems strange that structures so sensitive in this respect can undergo extreme structural changes without giving rise to pain; but, so far as is known, arteriosclerosis may transform the elastic and muscular tissues of arteries into degenerated tissue containing plaques of calcium without producing pain. The walls of the veins, on the other hand, are much less sensitive to mechanical stimulation, and the pain apparatus in them is evidently less complex than that of arteries.

Muscles form an interesting comparison with other tissues, for while there can be no doubt that they have a well organized pain apparatus, it is usually

excited by a special form of stimulus, i. e., spasmodic contraction as in cramp, or by contraction with impaired blood supply.

Pain is commonly regarded as a protective mechanism and therefore primitive in character, but it is a more highly specialized sense than would at first appear. The corium, lying under the tactile epithelium of the entire surface of the body, is a layer of tissue supplied with nerves responsive to a wide range of stimuli, which acts as a protector against many injurious agencies. Pain is not produced by nerves of other forms of sensation, but has its own apparatus, its own nerves and probably its own receptors.

BECK, Buffalo.

CONCENTRIC SCLEROSIS AND THE PHYSICOCHEMICAL FACTORS IN THE DEVELOPMENT OF PROCESSES OF DEMYELINIZATION. J. HALLERVORDEN and H. SPATZ, Arch. f. Psychiat. **98**:641 (Feb.) 1933.

Hallervorden and Spatz discuss concentric sclerosis on the basis of two cases of their own and four that have been reported by others. The material of three of the latter was reexamined by the authors in connection with their own cases. This disease process was first described by Baló under the term of "leukoencephalitis concentrica," which belongs to a group that includes also multiple and diffuse sclerosis. The general characteristic of all of them is demyelinization. Clinically, the picture does not differ essentially from that of diffuse sclerosis. Anatomically, it is characterized by the development of concentrically arranged layers of demyelinization, forming foci in the white matter of the cerebrum separated from one another by areas of preserved myelin. The focus develops around a blood vessel, the oldest layers being next to the vessel wall while the more recently developed layers are at the periphery. Histologically, the layers of demyelinization present a structure identical with that in the foci of multiple and diffuse sclerosis. The authors are of the opinion that the foci are caused by a special substance ("lecitholytic ferment" of Marburg) which has a particular predilection for the myelin sheath and enters the brain tissues by virtue of a disturbance in the blood-brain barrier. The diffusion probably occurs in rhythmic fashion so that it leads to the development of concentric layers in a manner not unlike the development of the rings of Liesegang. The development of different layers is due probably to repeated interactions between this invading agent and the antibodies produced by the brain tissue. The difference between concentric sclerosis, on the one hand, and multiple and diffuse scleroses, on the other, is not quite as sharp as has hitherto been thought, for one finds not infrequently ringlike formations in the latter and irregular foci in the former. The reason for the predominance of irregular patches in multiple and diffuse scleroses is probably to be sought in a different type of diffusion of the myelolytic substance. In multiple sclerosis the diffusion probably comes from the blood vessels, whereas in diffuse sclerosis it comes from the fluid within the ventricles. The authors do not offer any suggestions as to the etiology of the development of this substance, but they are of the opinion that it can probably be traced either to the toxin of some infectious agent or to some product of metabolism.

MALAMUD, Iowa City.

A CASE OF HEMIATROPHY OF THE FACE WITH AUTOPSY OBSERVATIONS. A. STIEF, Ztschr. f. d. ges. Neurol. u. Psychiat. **147**:573, 1933.

This is said to be the first case of facial hemiatrophy in which an autopsy has been performed. It concerns a woman, aged 60, who was left-handed; there was progressive atrophy of the right side of the face, which later spread to the right side of the throat and shoulder and the right lower limb. At the beginning the condition was considered to be idiopathic hemiatrophy, but in view of the further course with severe dementia and neurologic manifestations (pain in the face, atrophy of the soft palate on the right, rigidity of the right side and an occasional Babinski phenomenon) a diagnosis of symptomatic hemiatrophy was made. At autopsy the atrophy was seen to involve the deeper structures and inner organs. Clinically

there were evidences of right-sided sympathetic disturbance (increase of the glandular structures on the right and Horner's syndrome, with characteristic changes of the pupil on administration of atropine, cocaine and epinephrine). At autopsy the pleura was adherent to the right lung. Histologically, there were marked disturbance of the right inferior cervical ganglion and severe infiltration in the superior cervical ganglion while the left sympathetic nerves showed no changes.

The histopathologic diagnosis of the conditions in the brain was arteriosclerosis cerebri with severe circulatory disturbances, which differed in the two hemispheres. On the right there were dilatation and stasis; on the left there was rather a narrowing of the terminal vessels. As a result of these divergent circulatory changes the parenchymatous disturbances were different. The changes in the vessels involved the whole hemisphere and not distributions of single vessels. There were no mechanical closures of any of the large vessels. Stief considers that all these observations are evidences of the true rôle of the sympathetic system in relation to the pathogenesis of the circulatory changes.

In relation to the pathogenesis of the hemiatrophy, Stief believes that, besides the degeneration in the contralateral hypothalamic region, one can also postulate a simultaneous homolateral disturbance of the sympathetic system.

WINKELMAN, Philadelphia.

EXPERIMENTAL CONVULSIONS. S. BERNARD WORTIS, Am. J. Psychiat. **11**:611 (Jan.) 1932.

Using a 10 per cent solution of camphor monobromide in alcohol, Wortis was able to produce convulsions in cats by injecting this solution into the femoral vein. Normal animals required about 0.02 cc. of the preparation per pound of body weight. Removal of the frontal area of both cerebral cortices did not alter the animal's sensitiveness to the convulsant. When the left motor cortex was ablated, however, the convulsions elicitable within a week after the operation were clonic in the left limbs and tonic in the right. Removal of the parietal and occipital cortex without injury to the underlying thalamic region did not alter the cat's response to the camphor. Ablation of the left cerebellar lobe was followed by no changes in response to the convulsant. When left striatal lesions were experimentally produced, the convulsions occurred after injection of camphor, except when the motor cortex or pyramidal tract had been injured.

Decerebration was produced by an incision from the rostral border of the superior colliculi through the optic chiasm. Within four hours after operation the cats were able to walk about. Injection of camphor monobromide immediately or within twenty-four hours failed to provoke the convulsive response, although tonic extension of the limbs obtained, accompanied by marked respiratory stimulation.

Cervical sympathectomy failed to protect the animal against seizures. The introduction of foreign bodies, some occupying as much as 4 per cent of the intracranial volume, into the cranial cavity did not alter the responses, except when the motor cortex was directly pressed on. In the latter cases the covulsion was tonic on the contralateral side.

DAVIDSON, Newark, N. J.

RECTAL PARALDEHYDE BEFORE OPERATION. J. DUKE STEWART, Brit. M. J. **2**:1139 (Dec. 24) 1932.

Stewart records his experiences in a series of 500 cases in which paraldehyde was administered rectally before operation. The paraldehyde is given in solution with saline solution, well shaken together, in the proportions of 1½ ounces (44.3 cc.) of saline solution to 1 drachm (3.7 cc.) of paraldehyde. The average adult dose of paraldehyde is 8 drachms (30 cc.). If the patient's weight is less than 112 pounds (50 Kg.), the amount is diminished. It should be administered slowly and is then retained without difficulty. The paraldehyde used must be fresh. In the author's opinion, paraldehyde administered rectally provides a safe and certain method of

eliminating for the patient the more unpleasant accompaniments of operation. Sometimes other anesthetics are unnecessary, but when they are used, they are administered without difficulty; usually a very small amount is required. Spontaneous awakening generally occurs from five to eight hours after the operation, but the patient remains drowsy and comfortable for a much longer period. In most cases he secures a very restful first night, often without the aid of morphine. It is of particular value with a nervous patient. Postoperative vomiting appears definitely to be reduced. In respect to safety and certainty of action, paraldehyde premedication appears to compare favorably with that effected by such drugs as scopolamine, pentobarbital sodium, phenobarbital and avertin, and it is not accompanied by the fall of blood pressure and other dangers associated with avertin. Disadvantages are, for the most part, the unpleasant odor of paraldehyde appearing in the breath shortly after the rectal administration; frenzy and excitement may occur in a small number of cases. This, however, is always of short duration and is of little significance.

FERGUSON, Niagara Falls.

A CONTRIBUTION TO THE VERTEBRAL MEDULLARY MANIFESTATION OF STATUS DYSGRAPHICUS. P. OTTONELLO and G. BIGNAMI, Riv. di pat. nerv. **40**:36 (July-Aug.) 1932.

The authors present four cases in which neurologic conditions were associated with vertebral malformations. In two, in which vertebral malformations were associated with syringomyelia, the authors favor the embryonal origin of syringomyelia proposed by Bielschowsky and Unger, according to whom there are sufficient reasons to exclude postfetal causes for the development of syringomyelia.

The authors also stress that in association with vertebral and medullary malformations embryonal malformations may exist in other organs. Thus, there may be malformations of the kidney, the ureters or the bladder. The presence of an occult spina bifida is more rare. Spina bifida may be the expression of a disturbed morphogenesis, the results of which are seen not only in the vertebral column but also in other districts of the body which have regional embryogenic relations with the abnormal vertebral section. Altogether, the findings of vertebral malformation and involvement of the corresponding medullary segments would point to the unity of the causative agent exerting its influence in embryologic life on both the bony and the nervous structures. Tumor-like masses at times develop along the posterior line of suture of the spinal cord and present histologically a blastomatous appearance; the authors think that the disturbing factor leading to the growth in these cases must act in a very early phase of ontogenetic development, during the stage of migration of the ependymal cells. The cause of such perturbed development, resulting in malformation of the bone, is still unknown, and the authors make no attempt to dwell on the various complex mechanisms which might lead to that final result.

FERRARO, New York.

THE EXPERIMENTAL METHOD TREATED AS AN INSTRUMENT OF PSYCHOLOGICAL INVESTIGATION. ERNST KRETSCHMER, Charact. & Personal. **1**:175 (March) 1933.

In their attitude toward the experimental technic, psychologists have shifted from the reverential overemphasis of a generation ago to the almost contemptuous tolerance of today. Yet neither extreme estimate is justified; experimental technic enjoys the same merits and suffers the same limitations in psychology as in other sciences. Kretschmer studied the problem of the relationship between the physical and psychic manifestations of personality by both approaches, the descriptive and the experimental, and found the results in close agreement. The descriptive method gave a more complete and more real, if somewhat less accurately defined, understanding of the problem, while the experimental technic, although affording an irrefutable degree of factual pre-

cision, was able at its best to isolate only a small number of factors from the complex totality. These differences exist, however, in all sciences. In the realm of physics, for example, the observer does not, strictly speaking, measure heat, but rather the expansion of a column of mercury or the amount of absorption of energy, the latter being again measured indirectly. So in psychology, an emotion may be described by the subject or perhaps sensed by the observer, while it may be measured by the psychogalvanic changes or the alterations in the respiratory curve. Both methods must be used, each complementing the other and demanding interpretation by a trained observer. Deprived of the descriptive method, psychology would lose much of its richness and completeness, while without the experimental approach it would be robbed of preciseness.

DAVIDSON, Newark, N. J.

PRISON PSYCHOSES IN WOMEN. F. KNIGGE, Arch. f. Psychiat. **97**:533 (Sept.) 1932.

Knigge presents a study of 60 women with mental disease who were sent to the hospital from various prisons. This is a continuation of a previous report in which a similar investigation was undertaken of 640 men with mental disease. Of the 60 patients, 44 were serving sentences for various crimes, and 16 were in prison awaiting trial. The psychoses either developed or became obvious during confinement in prison. Four patients were suffering from dementia paralytica, 3 from cerebral syphilis; 8 had schizophrenic psychoses; 4 had psychoses due to previous misuse of drugs; 1 had a psychosis with hyperthyroidism; 1 had a manic-depressive psychosis, and 39 cases belonged in the group of so-called psychogenic prison psychoses. The small number of women as compared with men in whom psychoses developed is partly explained on the basis of the smaller number of women in the total prison population and the relatively smaller incidence of psychopathic personalities in female prisoners as compared with males. The psychoses, outside of those diagnosed as psychogenic prison psychoses, did not show any definite differences from those in patients with the same mental diseases in the general population. The actual prison psychoses in women seemed to be characterized by a relative preponderance of explosive emotional reactions and tended to show more of an erotic coloring than is the case in male patients. Contrasted with this is the relative absence of prison disturbances characterized by psychic defense reactions and by paranoid litigation tendencies.

MALAMUD, Iowa City.

VARIATIONS OF PUPILLARY TONUS: STUDY OF THE PHOTOMOTOR PUPILLARY REFLEX BY TRANSPALPEBRAL LUMINOUS PROJECTION. PAUL MORIN, Rev. d'oto-neuro-opht. **10**:721 (Dec.) 1932.

Differences in the size of the pupil may best be observed in convergence, and better synergy in convergence is obtained when the patient is asked to fix on his own finger placed on the upper part of the back of the nose below its root. In order to eliminate disturbing factors (accommodation and the emotional element), examination in a dark room with graduated intensities of light and in different positions of the globes was undertaken. It was observed that lateral deviations of the globe caused dilatation; convergence produced contraction; upward deviation resulted in dilatation, and downward deviation was accompanied by contraction. Inward deviation without convergence produced contraction. Morin believes that these phenomena are due to modifications of pupillary tonus, and that there is a close relationship between the center for pupillary tonus and the nuclei of the common motor oculi nerve. A study of several methods of examination has resulted in the conviction that transpalpebral illumination yields the most accurate and constant results. The technic is to apply a light of moderate intensity (pocket flashlight) to the closed eye and to observe the contralateral reflex. It is essential that the light be transmitted through the pupil. The patient is seated; the eyes

are turned slightly upward, and the light is applied to one of the closed lids. At the moment the other eye is opened the light is switched on and the pupil is observed.

DENNIS, Colorado Springs, Colo.

HUGHLINGS JACKSON'S OPINIONS ON EPILEPSY. O. R. LANGWORTHY, J. Nerv. & Ment. Dis. **76**:574 (Dec.) 1932.

Despite the fact that he published his paper fifty years ago, the ideas of Hughlings Jackson are still appreciated for their ruggedness and profundity. Little additional information as to the physiology of convulsive seizures has been added since. Jackson formed the hypothesis of three levels of function in the central nervous system. The lowest level included all motor cells and fibers below the level of the cerebral cortex. The next level was the so-called motor cortex of both cerebral hemispheres. The third level included all parts of the cerebral cortex aside from the motor area. The seizures originating at the highest level in the great association areas of the cortex are generalized from the beginning, and are of the type of the so-called idiopathic epilepsy. Hughlings Jackson defined epilepsy as the name for occasional sudden, excessive, rapid and local discharges of the gray matter. He was the first to believe that the convulsion originated from the cortex, and reiterated the theory of Laycock that the cerebral cortex was subject to reflex action. Convulsions were due, according to Jackson, to the storing up in the cerebral cortex of an excess of energy in unstable equilibrium. The localized or jacksonian convulsions he recognized as caused by demonstrable lesions in the region of the motor cortex. He also accumulated evidence to show that there are centers for the control of the autonomic nervous system in the cerebral cortex.

HART, Greenwich, Conn.

TEMPORARY VISUAL DISTURBANCES AS AN INITIAL SYMPTOM OF DIABETES MELLITUS. H. P. HIMSWORTH, Brit. M. J. **2**:1184 (Dec. 31) 1932.

This study is based on 100 cases of diabetes mellitus, in which each patient was examined specifically for disturbances of vision. In each case the eyes were examined by the retinoscope and the ophthalmoscope. All patients showing gross organic damage such as cataract, retinitis and hemorrhages into the vitreous were excluded. The cases were divided into two groups, those occurring in persons under 45 and those in patients over that age. In the first age group, a history of temporary visual disturbance was given in 49 per cent of the cases, and in the second group, in 21 per cent of cases. Thus ,temporary visual disturbances, apparently referable to changes in refraction, have occurred as an initial symptom in 34 per cent of these 100 cases of diabetes. All cases had this in common: the derangement of vision was only temporary. The symptom has never been noted in the absence of marked glycosuria. The symptom has never been reported in cases of glycosuria which on investigation have been found not to be cases of diabetes. In eight proved cases of renal glycosuria it was absent.

Thus, it seems permissible to conclude that rapid changes of refraction are in themselves sufficient for a diagnosis of diabetes mellitus, though this deduction could not be securely accepted unless it was fully proved by many observers that similar visual disturbances are never found in other diseases.

FERGUSON, Niagara Falls, N. Y.

SYMMETRICAL ATROPHIC NECROSIS OF THE ASCENDING PARIETAL AND THE OCCIPITAL CONVOLUTIONS. G. GUILLAIN and I. BERTRAND, Ann. de méd. **31**:35, 1932.

The history is reported of a woman, aged 58, who, following a cold, suffered from intensive headaches for three weeks. These were followed by diplopia, bilateral ptosis and intensive pain in the right arm, which became totally paralyzed. Involuntary athetoid movements of the left arm followed, with intensive pain. Five years later she was completely blind (only vision of both maculae was pre-

served). The fingers of the hand were flexed, with the exception of both thumbs, which were extended. There was a diminution of sensation for touch, pain and temperature in parts of the hands and lower arms, with complete absence of proprioceptive sensation and with astereognosis in both hands. Following an apoplectic attack with left hemiplegia in the twelfth year of the disease, the athetoid movements disappeared, and the fingers of the left hand were more easily moved and were less spastic than before. She died three days after the apoplexy.

The clinical diagnosis of a bilateral thalamic syndrome was not confirmed by the autopsy. Both thalamic nuclei were grossly intact. There was a bilateral symmetrical destruction of both occipitoparietal regions, with partial involvement of the precentral gyri. The softening was most pronounced in both occipital poles. The case was reported in order to demonstrate that "a thalamic syndrome" may also be produced by bilateral involvement of the cortical sensory area.

WEIL, Chicago.

THE FUNCTION OF THE ADRENAL MEDULLA. E. ANNAU, S. HUSZÁK, J. L. SVIRBELY and A. SZENT-GYÖRGYI, J. Physiol. **76**:181 (Oct. 4) 1932.

Fresh suprarenal medulla contains, under certain conditions, a substance having a more potent pressor action on the blood pressure, and a more potent inhibitor action on the isolated intestine than epinephrine. If small doses of epinephrine are compared with active extracts containing colorimetrically the same amount of epinephrine in the decapitated cat, a much stronger pressor response will be given by the latter. If the dose of extract is gradually decreased, the dose of epinephrine being kept constant, from one tenth to one fifteenth of the dose of extract will give a response equal to that given with epinephrine. The same is true in cats after decerebration or under ether anesthesia, provided the animal does not give a depressor response which makes the results incomparable. If the effects of big doses of epinephrine, producing a rise of blood pressure to the maximal height, are compared with those of an extract giving an identical color reaction, the response to the latter will show a much longer duration. If active extracts are compared with epinephrine for activity on an isolated loop of rabbit's intestine the inhibitor action of the extract will be found to be much stronger. Equally strong inhibitor actions are produced by epinephrine and by the extract when the latter contains from 10 to 15 times less epinephrine as measured colorimetrically. In the Trendelenburg frog preparation a similar difference is found in vasoconstrictor activity.

ALPERS, Philadelphia.

OBSERVATIONS IN CATATONIA WITH MIXTURES OF CARBON DIOXIDE AND OXYGEN. HAROLD KELMAN, Psychiatric Quart. **6**:513 (July) 1932.

Using a mixture of carbon dioxide (60 per cent) and oxygen (40 per cent), with a McKesson anesthetic machine, Kelman obtained good results in terminating temporarily the mutism of fifteen patients with catatonic dementia praecox. In all the one hundred and sixty-three treatments, there was only one instance of respiratory collapse, and there were no fatalities. The gas mixture was administered for from ninety to one hundred and twenty seconds, the mask being removed when respiration became irregular, heart action poor, pupils widely dilated or the extremities limp. Some of the patients enjoyed the sensation so much that they wanted the procedure repeated immediately. The physical changes noted during the inhalation were: twitchings, tremors, mydriasis, tetany, transitory exophthalmos and transitory prominence of the thyroid gland. Patients became lucid within three minutes after removal of the mask, and talked or answered questions relevantly for from four to twenty minutes. It was often possible to obtain from the subject a coherent account of the development of the psychosis during a series of lucid intervals. Favorable results were noted in younger patients (under the age of 28), treated early in the course of the stupor, possessed of good intelligence and of not too typical a schizoid personality type. Clinical improvement was noted in thirteen of the fifteen patients, and in one case recovery seems to have been complete.

DAVIDSON, Newark, N. J.

THE FATTY SUBSTANCES IN NEURINOMAS OF THE ACOUSTIC NERVE: HISTOPATHOLOGIC AND HISTOCHEMICAL RESEARCHES. UMBERTO DE GIACOMO, Schweiz. Arch. f. Neurol. u. Psychiat. **31**:73, 1933.

This paper presents, in addition to a review of the pertinent literature, the results of exhaustive histopathologic and histochemical studies of the fatty changes in two acoustic neurinomas. The fat was found lying free in the tissue in the form of tiny droplets, as well as enclosed within the so-called xanthomatous cells. The free droplets seemed to have a predilection for the fibrillar portions of the tumor in which they tended to group themselves about the nuclei, some of which showed certain regressive changes. The droplets exhibited a further predilection for areas in which the nuclei were relatively sparse. Fat-containing xanthomatous cells scattered throughout the substance of the tumor appeared to have extended out from collections of similar cells grouped about blood vessels and beneath the capsule. De Giacomo expresses the view that the fat droplets were the end-products of degenerative protoplasmic changes resulting from mechanical and circulatory effects of the growth of the tumor within its capsule, and that these droplets were ingested by the xanthomatous cells which, he believes, were of mesenchymal origin. Both the free droplets and the intracellular collections were well defined chemically and appeared to be unsaturated phosphatides rather than sterols.

DANIELS, Denver.

CIRCUMSCRIBED TORSION SPASM. G. ROASENDA, Riv. di pat. nerv. **40**:112 (July-Aug.) 1932.

Roasenda reports eight cases of localized torsion spasm. Four developed after epidemic encephalitis. Another presented the spasm in association with tremors that were not related to encephalitis. The sixth case presented a torsion spasm localized in the left arm and left foot, and was correlated with a cerebral circulatory disturbance. The seventh case was of undetermined origin, and the eighth may possibly have developed as a result of an encephalitic process in the mother during pregnancy. In one case of encephalitic origin the manifestations in the lower extremities were similar to those in case 6, related to cerebral circulatory changes. Case 4 was particularly interesting because of the concomitance of several associated movements (tremors, myoclonias, torticollis and other movements). As to the pathology of the condition, Roasenda thinks that the putamen is often involved, and that the globus pallidus and mesencephalon, particularly the red nucleus and the corpus Luysii, also participate in the process. Roasenda is of the opinion, already expressed by Jakob, that torsion spasm is a symptom complex and not a clinical entity. It seems that some cases are familial and that Russian Jews are particularly subject to the disease.

FERRARO, New York.

VENOUS AND ARTERIOVENOUS ANGIOMAS OF THE BRAIN. A CLINICAL AND ROENTGENOGRAPHIC STUDY OF EIGHT CASES. S. BROCK and C. G. DYKE, Bull. Neurol. Inst., New York **2**:247 (July) 1932.

The authors report three cases of venous and five cases of arteriovenous angiomas of the brain. The study deals mainly with the clinical features of these conditions. Extracranial vascular lesions existed in four cases. An unusual case of venous angioma of the retina, chiasm, midbrain and cerebellum is included. The important ocular signs were homonymous hemianopia and unilateral exophthalmos. The arterial bruit of extracranial or intracranial origin was also an important sign; it is frequently overlooked. Roentgen findings were of great value, showing intracerebral calcification and dilatation and tortuosity of the vascular channels. The peculiar character of the calcification in the venous angiomas is considered pathognomonic. In the arteriovenous variety the cardiovascular phenomena were of diagnostic significance. These include enlargement of intracranial, extracranial, and carotid arteries and of the heart. A systolic mitral murmur, a mild degree of

tachycardia, low systolic and a much reduced diastolic blood pressure were sometimes found. The article contains a number of excellent plates and sketches. Literature on the subject is briefly reviewed and methods of treatment are discussed.

KUBITSCHEK, St. Louis.

COMPLETE SYNDROME OF THE LAST FOUR CRANIAL NERVES (COLLET) WITH PARALYSIS OF THE SYMPATHETIC OR SYNDROME OF THE PAROTIDEAN POSTERIOR SPACE (VILLARET). G. GALAND, J. de neurol. et de psychiat. **32**:723 (Oct.) 1932.

Galand reports the case of a boy in whom, following an injury to the skull which did not cause unconsciousness, there developed a cervical tumor on the right side and a protrusion of the right tonsil, but no associated tonsillitis. After three days there was a discharge of pus into the throat; on the following day still more pus was discharged, with a fall in temperature. Two days later, there was hematemesis, which recurred in eighteen hours. On the next day there was a pupillary inequality, with myosis on the right side, and deviation of the tongue to the right; the uvula was drawn to the left. A few hours later, the pain and cranial nerve involvements were more marked and were associated with the oculocardiac reflexes, and eventually with complete paralysis of the right vocal cord. This cranial nerve syndrome was first described by Collet after an injury in war. During the first few days hematemesis was the apparently important symptom, but this resulted from blood swallowed from the wound in the posterior pharyngeal wall, the hemorrhage coming from some vessels torn by the fracture of the skull.

WAGGONER, Ann Arbor, Mich.

THE FUNCTIONS OF THE GREAT SPLANCHNIC NERVES. D. T. BARRY, J. Physiol. **75**:489 (Aug. 10) 1932.

A method was adopted for investigating the functions of the great splanchnic nerve in the dog and rabbit by splitting the trunk longitudinally into two or three branches and stimulating each separately. The method is not altogether a satisfactory one, but the results afford certain indications that these functions are more complex than is generally supposed. Stimulation of a branch sometimes caused a fall of blood pressure, accompanied by inhibition, augmentation or no change of intestinal movements. Stimulation of a branch sometimes occasioned greatly increased activity of intestinal movement, with or without preliminary inhibition. This reaction is similar to that obtained by stimulation of the vagus nerve, but there is no evidence that the parasympathetic system is in play. The results, as stated, have been found chiefly in the dog and not satisfactorily in the rabbit. In the dog the great splanchnic nerves contain hypotensive vascular fibers and augmentor intestinal fibers which, with what is generally admitted to be the fiber of opposite function, in each case constitute two reciprocal systems.

ALPERS, Philadelphia.

PROBLEM OF TRAUMATIC PARKINSONISM. A. E. KULKOV, J. Nerv. & Ment. Dis. **75**:361 (April) 1932.

Two cases are presented in which trauma is considered as a predisposing factor in the origin of the disease, accompanied at the same time by rudimentary epidemic encephalitis. This association of trauma with encephalitis has been pointed out by Crouzer and Latmar. Maier declares that patients complaining of traumatic neurosis are suffering from a previous encephalitis. During the war, many cases of cerebral trauma were reported, but no cases of paralysis agitans resulted. Bing considers that the trauma may activate the virus of the disease but always plays a secondary rôle. The author does not think that the rôle of the trauma as a primary or secondary factor has yet been settled.

HART, Greenwich, Conn.

DIAPHRAGMATIC TIC RELIEVED BY SECTION OF PHRENIC NERVES; REPORT OF TWO CASES. HUGH SMITH, Am. J. M. Sc. **188**:837 (June) 1932.

In the first case reported, the history was suggestive of a possible relationship between repeated intestinal obstructions and the diaphragmatic tic. The right phrenic nerve had to be sectioned twice in twenty-two months, the left twice within a three day period; the patient experienced five attacks of intestinal obstruction within a period of thirty-nine months. The second case was that of a diaphragmatic tic following a definite attack of epidemic encephalitis. The right phrenic nerve was sectioned, with temporary relief, but with regeneration of the nerve in three months there was a reappearance of the tic. Previous observations of Dowman and Gamble and others are corroborated. MICHAELS, Boston.

VAN DER HOEVE'S SYNDROME. HUGUES, P. CAZEJUST, H. VIALLEFONT and A. RATIÉ, Rev. d'oto-neuro-opht. **10**:693 (Nov.) 1932.

A patient with a schizophrenic syndrome, with beginning melancholia, was found to have a deep blue color of the sclera. It was not possible to examine the patient satisfactorily, but it was determined that she was deaf, and that there were an ununited or fractured olecranon process and relaxations of the ligaments of the fingers. The family history was unimportant, except that the father became deaf following a fall at the age of 7 years. Laboratory examinations revealed a positive (+) Bordet-Wassermann reaction, hypophosphoremia and hypercalcemia.

DENNIS, Colorado Springs, Colo.

A CASE OF CONGENITAL ELEVATION OF THE CERVICAL SPINE ASSOCIATED WITH SYRINGOMYELIA. FELIX DU TOIT, Brain **54**:421 (Dec.) 1931.

The case reported is the second with a special neurologic condition to be described. Marked elevation of the left scapula, shortness of the neck, tilting of the head and scoliosis were the most conspicuous features of the deformity. Sprengel believed that etiologically the deformity was the result of malposition of the fetus in utero, but generally it is attributed to an arrest in development. A Klippel-Feil syndrome (fusion of the cervical vertebrae) was also present. The neurologic disturbances seemed to point to syringomyelia. MICHAELS, Boston.

LIPOID HISTIOCYTOSIS (NIEMANN-PICK TYPE). SHERL J. WINTER, Am. J. Dis. Child. **43**:1150 (May) 1932.

The author reports two cases of Niemann-Pick disease in sisters. In the first case there was no autopsy; in the second, an autopsy was done, but no report was made concerning a lipoid involvement of the brain. Conglomerate tuberculosis of the apex of the lung and of the bronchial lymph nodes was discovered in the case in which autopsy was performed. The author reports that the cases were essentially typical of the disease. The interest of the cases centers in the familial incidence.

LEAVITT, Philadelphia.

TUBEROUS SCLEROSIS. HAROLD L. STEWART and E. L. BAUER, Arch. Path. **14**: 799 (Dec.) 1932.

The clinical and pathologic features of two cases of tuberous sclerosis are presented and discussed. One of the cases, which was complicated by adenoma sebaceum, occurred in a Negro infant. In obscure cases of obstructive or non-obstructive hydrocephalus associated with epilepsy, with or without adenoma sebaceum, tuberous sclerosis must be considered as a possibility. The features of lipodystrophy are emphasized, and it is suggested that tuberous sclerosis is a congenital metabolic disturbance.

WINKELMAN, Philadelphia.

CORTICAL REPRESENTATION OF VISION. GORDON HOLMES, Brain **54**:470 (Dec.) 1931.

An interesting clinical case of special visual disturbance with autopsy is presented. With a partial left homonymous hemianopia in which the blindness came up to the fixation points along the horizontal meridians, there was a considerable escape along the vertical meridians in the upper and lower quadrants. The clinicopathologic findings tend to confirm the author's previous conclusions that those portions of the retinas that lie along the vertical meridians are represented on the exposed mesial surfaces of the occipital lobes, and, by exclusion, that the retinas along the horizontal meridians are projected onto that part of the area striata which lies in the walls of the calcarine fissures.

SPASMODIC TORTICOLLIS: RESULTS OF REMOVAL OF FOCI OF INFECTION AND TREATMENT WITH SPECIFIC VACCINE. EDWARD H. RYNEARSON and HENRY W. WOLTMAN, Am. J. M. Sc. **183**:559 (April) 1932.

A study of eighty-two patients (forty-three males and thirty-nine females) who had received medical treatment for spasmodic torticollis is reported. The average age of the patients at the onset of the condition was 39 years. Thirty-five patients gave evidence of focal infection in both the teeth and the tonsils. There was a favorable difference of 15 per cent in the improvement or disappearance of the torticollis with the removal of foci. Conclusions as to the value of specific vaccine therapy could not be drawn.

REFLEX GRASPING ASSOCIATED WITH TUMOURS NOT INVOLVING THE FRONTAL LOBES. PAUL C. BUCY, Brain **54**:480 (Dec.) 1931.

Cases from the literature are reviewed to show that bilateral reflex grasping is of no certain value as a localizing sign. Two cases are described: one, a tumor of the fourth ventricle with a marked internal hydrocephalus, and the other, a large neoplasm in the right occipital lobe, in both of which bilateral reflex grasping was present. It is stressed that in the presence of marked internal hydrocephalus, with increased intracranial pressure, the value of reflex grasping as a localizing sign is greatly reduced.

THE RATE OF CONDUCTION IN THE HUMAN MOTOR NERVE. F. GOLLA and S. ANTONOVITCH, Brain **54**:492 (Dec.) 1931.

There are no consistent figures as to the velocity of nervous impulses. The results reported by previous investigators are discussed as to method. A special technic is described; no significant discrepancies of results were encountered. The conduction rate of the ulnar nerve is 38.7 meters per second, with a possible error of not more than 5 per cent. There is no reliable information as to the rate of conduction of proprioceptive impulses from tendon joints and muscles.

MICHAELS, Boston.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY AND NEW YORK ACADEMY OF MEDICINE, SECTION OF NEUROLOGY AND PSYCHIATRY

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CLARENCE P. OBERNDORF, M.D., *President, in the Chair*

EPILEPSY: TREATMENT OF INSTITUTIONALIZED ADULT PATIENTS WITH A KETOGENIC DIET. DR. JOHN NOTKIN.

This article was published in full in the April, 1934, issue of the ARCHIVES, page 787.

DISCUSSION

DR. IRVING SANDS: In 1919 I introduced phenobarbital as a therapeutic agent in cases of epilepsy at the Manhattan State Hospital (Control of Epileptic Seizures, *State Hosp. Quart.* **5**:236 [April] 1920). The results were most striking (Luminal Therapy in the Control of Epileptic Seizures, ARCH. NEUROL. & PSYCHIAT. **5**:305 [March] 1921). After reading the reports of pediatricians who made extensive claims for the ketogenic diet, I tried it in my own cases, but with rather disappointing results. Dr. Notkin seems to have been similarly disappointed. Regarding the diet in epilepsy, I made an empirical observation while treating epileptic patients at Ward's Island, namely, that they did much better when the amount of meats and meat soups was restricted. I still am convinced that phenobarbital is the best therapeutic agent for epilepsy.

DR. JOSHUA ROSETT: Dr. Notkin's report on the effects of a ketogenic diet in epilepsy is not favorable, nor is that of Dr. Sands. But the experience of a large number of reliable investigators with the administration of that diet to epileptic patients is that there is a diminution in the number and the severity of the attacks. No matter how good the results may be, however, the regimen does not impress me as a sensible one. The administration of a ketogenic diet is equivalent to the introduction of certain poisons into the patient's body; from a therapeutic point of view, it makes little difference whether they are introduced by the physician directly from the outside or indirectly by an abuse of the patient's metabolic powers. Whatever difference exists, however, favors the former, since it spares the patient the torments of a disgusting diet. Still worse than the ketogenic diet is the attempt to reduce the number of attacks by subjecting the patient to the torments of thirst.

While the relatively innocent barbital drugs and the bromides are at one's disposal it seems to me unnecessarily cruel to make the epileptic patient's life miserable by diets or by depriving him of water.

PROF. F. H. PIKE: Three questions arise in connection with this subject. The first concerns fat metabolism, the study of which has brought forth some surprising facts. The conclusion was reached before or during the World War that human beings could get along without fat, but it was found that they could not. One could not stop the war to find out whether the trouble was with the patients or with the theory.

The second question concerns the nature of the processes involved in the genesis of convulsions.

The third question is whether or not any modification of fat metabolism can influence these processes. Pediatricians think that it can in children. In older patients the results are not so good. An attempt has been made to study the problem experimentally.

The theory of the ketogenic diet does not seem to fit altogether with what is known about conditions that influence the actual excitability of nerve cells and nerve fibers. For instance, the phrenic nerve descends in contact with the pericardium, but it is not excited by the action current of the heart. Hiccups do not result. If, however, the concentration of carbon dioxide and of hydrogen ions is increased, the nerve responds to the action current of the heart. The diaphragm twitches with practically every contraction of the heart.

Somewhere between the results obtained by pediatricians with the ketogenic diet in children and the effects on the nerves of increasing the hydrogen ion concentration, and the results obtained with the diet in adults, there is some contradiction which apparently requires further investigation.

DR. JOHN NOTKIN: In reply to Dr. Pike, McQuarrie believes that the acid-base change which takes place while the ketogenic diet is being used is an incidental and not the responsible factor in the control of the attacks. He believes that the dehydrating effect of the diet has a favorable influence on the seizures (McQuarrie, I., and Keith, H. H.: Experimental Study of the Acid-Base Equilibrium in Children with Idiopathic Epilepsy, *Am. J. Dis. Child.* **37**:261 [Feb.] 1929. McQuarrie, I.: The Relationship of the Water Balance to the Occurrence of Seizures, ibid. **38**:451 [Sept.] 1929).

RÔLE OF THE FRONTAL LOBES IN INTELLECTUAL FUNCTION: A STUDY BASED ON A CASE OF PARTIAL BILATERAL FRONTAL LOBECTOMY. DR. RICHARD M. BRICKNER.

A right-handed man, aged 45, in August, 1930, was subjected to bilateral frontal lobectomy because of a tumor. Practically all of both lobes was removed, except the motor and premotor zones and Broca's area. Neither aphasia nor motor disturbance was evident following operation. A detailed psychologic study of the patient was made, which was used as a foundation for a theory concerning the function of the frontal lobes. The theory was compared with other views on the subject, some of which were similar.

In the present study, an attempt was made to clarify the subject by presenting the theory first. It was stated that if the theory were true, certain symptoms would be expected to result from bilateral frontal lobectomy. It was then shown that these were the patient's chief symptoms.

The theory, in brief, is as follows: Combined engrams of many varieties are formed in the occipital, parietal and temporal lobes (posterior association area of Flechsig). These combinations may reach a considerable degree of complexity. The frontal lobes (anterior association area of Flechsig) receive the engrammic combinations of the posterior association area and synthesize them into still more complex constellations. The latter are responsible for the most complex types of thinking and result in the formation of such faculties as those of judgment, abstract thinking, restraint over emotional impulses and sense of humor.

If the frontal lobes are removed, the capacity to make such complex syntheses should disappear. The symptoms following lobectomy should be of two main types: (1) defects in complex, synthetic functions the existence of which depends on the synthesis of simple engrammic units and (2) manifestations referable to the separate activation of simple engrammic units which formerly were synthesized.

In the first type, one would expect impairment of the faculties already mentioned. The lack of adequate restraint over emotional impulses would result in prominence of one symptom in particular—great freedom of expression of emotional drives. The ones most likely to reveal themselves would be those ordinarily requiring the most restraint—viz., ego aggrandizement, sexuality and perhaps reactions of hostility. Secondarily, the "character" or "personality" of the person would appear changed; from a descriptive standpoint, it might be proper to say that he had lost his "social" or "moral" sense.

In the second type, such a phenomenon as particularization might occur; the patient, having to choose between two or more engrammic processes when there

had been only one before because of synthesis, would be unable to select details skilfully and would be likely to give them all. The same separate, unsynthesized activation of engrams might produce distractibility, one process continually distracting the patient from another. Moreover, when the separate processes were few, monotonous repetition or perseveration of thought might occur; this would result from the poverty of connection of these simpler engrams with others, compared with the richness of interconnection of the more complicated engrams formed in intact frontal lobes. Such nonsynthesis would lead to trying experiences, and the difficulty of making adequate intellectual efforts might be so great that it would be too much trouble to try. Lack of interest in the use of the intellect might develop into a state of apathy.

Study of the patient under consideration as well as of some of his previously recorded productions shows that a single theme runs throughout—intact simple units of thought are unsynthesized into coherent, complex aggregations.

The patient has several other symptoms which are not adequately explained in this way—compulsions, the use of jargon and a relatively fixed euphoric mood.

FUNCTIONS OF THE FRONTAL LOBES: OBSERVATIONS IN TWO CASES OF UNILATERAL LOBECTOMY. JAMES C. FOX, JR., and WILLIAM J. GERMAN, New Haven, Conn.

The exact location of the resection in two cases of tumor of the frontal lobe was determined by a study of the cerebral topography at operation, by observation of the position of the silver clips as revealed by roentgenograms and by examination and measurement of the portion removed. In the first case these observations were corroborated by postmortem examination. In each instance a glioma was situated in the right frontal lobe. In the first case the lobe was resected along a line beginning 6 cm. anterior to the superior extremity of the fissure of Rolando and ending 3 cm. anterior to the junction of the rolandic fissure with the fissure of Sylvius. The section passed through the anterior horn of the ventricle and the rostral end of the corpus callosum. The right olfactory nerve was severed, and the right anterior cerebral artery was clipped above and below the corpus callosum. In the second case the line of resection was about 1 cm. anterior to the precentral gyrus, curving anteriorly in the region of the inferior frontal gyrus, in an effort to preserve the motor speech area. The section passed through the anterior horn of the ventricle, but the anterior cerebral artery was left intact. A narrow shelf of brain covering the floor of the anterior fossa was not disturbed.

Resection of the major portion of the right (nondominant) frontal lobe in the first case apparently did not produce the slightest interference with the patient's ordinary language capacity or with the normal activity of the processes registered in two intelligence tests; apparently skilful habitual left-handed movements were unaffected. However, it appears probable that the left-sided coordinations suffered a slight amount of slowing. It seems evident that the resection involved an area which is concerned with motor visual processes. The disturbance did not hamper the ability to read appreciably, but the patient suffered considerable loss in the more precise visual acts of placing points and judging distances. Unfortunately, these results cannot be compared with observations obtained before he became ill. With the exception of the initial period of euphoria, the patient's personality following lobectomy did not seem to be essentially different from that prior to his illness. There was no significant change in the degree of initiative or motor activity and no impairment of intelligence and judgment. His mood reactions were normal and had undergone no alteration. The presence of episodic attacks of fear preceding by a short time the occurrence of neurologic symptoms is of special interest. Both in his family life and in his work the patient responded emotionally and behaved as characteristically as before. Lobectomy neither speeded up nor "put the brakes" on his motor drive.

Resection of a large part of the right (dominant) frontal lobe in the second case resulted in transient left hemiparesis and complete aphemia for one week, followed

by slight permanent impairment of the motor component of speech. The patient's writing showed occasional mistakes. There was no loss of the ability to perform skilled movements with the left hand. There was no evidence of any significant change in the intelligence or judgment, but quantitative psychologic tests were not carried out. Memory was good for both remote and recent events, except for a circumscribed period of retrograde amnesia. Emotional relationships to the family remained unaltered, but no reliable data are available on the sexual adjustment. For two months there was definite euphoria with occasional evidences of emotional instability, periods of depression, absence of spontaneity and loss of initiative. This dearth of spontaneous motor response persisted but gradually became less noticeable. By the fourth month following lobectomy, the chief alteration in the personality consisted of an increased facetiousness; the patient was well aware of the uninhibited nature of this jocosity.

Personality changes of this type suggest the association of such functions with the dominant frontal lobe. This is in accord with the views of von Economo, who on the basis of cyto-architectonic and comparative anatomic studies concluded that the anterior portions of the frontal lobe are concerned with the motor manifestations of the personality. "In the frontal lobe motility lies eccentrically in such a manner that the most simple motility occupies the precentral gyrus but it progressively increases in complexity and in psychic components in approaching the pole." However, one must be cautious in adopting any schematic conception such as this. For example, the tendency of a person to withdraw from social contacts may represent the natural reaction to a speech disorder. The dysphasic patient often "wise-cracks" in an attempt to cover up and distract attention from the underlying defect. With the loss of any specific function of the brain, various mechanisms are set into play and new domains of integration utilized, which employ whatever material is still available.

DISCUSSION ON PAPERS BY DR. BRICKNER AND DR. FOX AND DR. GERMAN

PROF. ROBERT S. WOODWORTH: The expectation may be that a psychologist much impressed by the work of his fellow psychologists Franz and Lashley will see in these reports fresh evidence for equipotentiality and mass action in the hemispheres. No doubt some sort of a case could be made out for the application of these conceptions. I have never been much impressed by them, because they seem to overlook the structural differences between different parts of the cortex. It seems to me that C. J. Herrick's recent work set that matter in a clear light.

With regard to the frontal lobes, which present an intriguing problem for the psychologist as well as for the neurologist, it seems to me possible to combine and harmonize the different views. There is the view of von Economo, mentioned by Dr. Fox, which attributes higher motor functions to the frontal lobes. There is the old view of Wundt, who laid emphasis on inhibition as the peculiar function of these lobes. One can classify under the head of lack of inhibition many of the symptoms reported by Dr. Brickner, such as the free expression and labile associations of his patient. Even the euphoria, the hypomania and the cheerful disposition in the face of an obviously serious situation may be conceived as being due to a lack of normal inhibition. However, it will probably be agreed that inhibition is not an ultimate explanation. There must be some positive activity that causes the inhibition, and here one may bring in the conception of synthesis stressed by Dr. Brickner. When the "synthetic function" is weakened, partial activities that should be held together escape from control, and so a deficiency of inhibition makes its appearance.

The function of the frontal lobes should be considered in relation to their anatomic position. Why should these functions of synthesis, control and inhibition be related to the part of the brain that abuts on the motor area and that may be considered an expansion of that area? Psychology suggests that the higher reaches of synthesis consist in what is commonly called voluntary action. Action appears to be the highest type of synthesis, while at the same time it is built on

bodily movement. In the realm of speech there are the synthesis of simple articulatory movements into spoken words, the higher synthesis of words into sentences and at a still higher level the organization of connected discourse, such as Dr. Brickner found lacking in his patient's conversation. The patient had many ideas to express, but lacked the power of organized expression. This conception of the function of the frontal areas in relation to speech is old, though I do not know to whom to credit it. In other spheres besides language, it seems to me, one can carry out the same conception of synthesis, namely, that it is an organization of action and of motor behavior guided, of course, by perceptions of the environment. The frontal lobes, superimposed on the motor region, would therefore be concerned in acting according to a plan, and the impairment of this function would show itself in relatively planless and heedless behavior.

DR. PAUL SCHILDER: I think that one generally asks whether a psychologic disturbance is in the intellectual or in the emotional sphere. There has been some justification for the distinction. But one should not forget that after all this separation of the emotional and the intellectual is artificial. Human beings are emotional as well as intellectual. There are no purely intellectual functions. There is an emotion in every thought; not only that, there is also motility. The whole personality is always involved in a multiplicity of functions. Therefore, the distinction between an emotional and an intellectual disturbance is more or less artificial, especially when one studies cases like the one which Dr. Brickner has presented so well. One sees immediately that there is not only a disturbance in intellectual synthesis.

I think that what is going on in the emotional sphere of this patient is more important than what is going on in his intellectual sphere. The patient jokes. One laughs at his jokes. There is an emotional contact between him and the listener. The emotional contact would not be possible if the emotional life of the patient had not been fundamentally changed. Of course, when the emotional life is altered, the aims, the strivings and the goal of the person are changed; life is easy; he does not ask so much of himself. A normal person has a plan; he lives in the schematic anticipation of what he expects. He is satisfied before he has reached his goal through trial and error, and then he compares the schematic anticipation with the actual result of his thinking processes. A patient with a lesion of the frontal lobe is much more easily satisfied than a normal person. He has not the same impulse to go further, owing to a fundamental change in his whole personality. When the case is considered from this point of view, there will not be any particular difficulty in understanding the euphoria of Dr. Brickner's patient. It is not something which is added. It is a part of his optimism, his self-contentment. The patient has changed his goal with the alteration in the total personality, especially in the sphere of the emotion. The patient was quick in his retorts, and often I had the feeling that it was more important for him to give a quick answer than to go deeper into the matter which he was discussing. It is true that this is an intellectual change. One may ask, what does such a change in reaction indicate? The patient seems to conclude his thinking processes more quickly than a normal person; he does not take time to "finish up" his thought. In other words, the frontal lobe is necessary for the consummation of thinking. I think that this general idea is of importance for the understanding of all cases of lesions in the cortical region.

I shall turn from this more or less general formulation to the more specific one, viz., that cortical lesions of different localization are concerned with the finishing, the ending processes of the different provinces of psychic life. The cases which have been presented show this particularly well. I am strongly opposed to the point of view which has been offered by Head, Goldstein and Lashley that the same disturbances are found in every case of cortical lesion. There is a sharp localization, and the cases which have been presented are particularly valuable from this point of view.

I have the following ideas about the function of the frontal lobe: Immediately before the motor area lies a region which is concerned with the finer elaborations

of the motor processes and impulses. In the motor aphasias, speech is not alone disturbed; the motor impulses involved in every motion (*Antriebe*) are also deeply affected. In lesions of this kind are found the akinesias. It seems that the parts nearer the frontal pole have something to do with the *Antriebe,* the impulses involved in the finer psychic function, the aims and goals of the person. Such a conception may be of importance to the general psychologist. I am firmly convinced that one will understand psychology much better from a study of these cases. The personality is striving, but the striving is altered. There is a changed attitude toward the world, and this change of attitude is certainly brought about by the lesion of the frontal lobe.

DR. CLARENCE P. OBERNDORF: In Dr. Brickner's interesting paper the opinion was presented that perhaps the frontal lobes assume the function of the superego of the Freudian terminology; in other words, that here is the location of the function of restraint or repression and inhibition. I agree with what Dr. Schilder said about emotion and intellectuality always moving in harmony .

Some one said that the superego is that part of the personality which "dissolves" in alcohol. Dr. Brickner's patient showed this type of expansiveness—perhaps a lack of the proper appreciation of the situation and a certain amount of flippancy, with all the keenness which a man shows when he is slightly under the influence of alcohol, when he has had just enough to make him alert and jocular but not foolish. If that is so, Dr. Brickner has demonstrated where alcohol acts when it removes the inhibiting functions of the mind. But I do not think that this theory is correct. I doubt whether there is any location in the mind in which alcohol acts specifically to remove inhibitions. Psychoanalysts have always hoped that in some way there would be a bridge between the physiologic and the psychologic. If such studies as those presented could show that the frontal lobe possesses the inhibiting function of the mind it would be of the greatest significance. The outstanding feature of Dr. Brickner's case is that the extraordinary degree of mental and emotional integrity which the patient exhibited could exist after the removal of so great an amount of brain tissue on both sides. It seems to me that all the evidence points to the fact that the mind works not in "localities," but rather as a whole. In all probability the functions of the supposititious superego are not localized in any one region of the brain.

DR. LEO DAVIDOFF: I wish to say a word about the question of the shift of cerebral dominance that Dr. Fox touched on. It is generally assumed, and is proved by at least one of his cases, that early training continuing for many years results in a shift of dominance from the left to the right hemisphere; that this is not always the case was illustrated by a recent unusual experience which I should like to cite. A man, aged 18, had been thrown from his perambulator at the age of 1½ years and sustained an injury to the head which resulted in right hemiplegia. He was of right-handed stock and had apparently attempted to use his right hand previous to the injury. After the onset of the right hemiplegia he became left-handed. Convulsive seizures set in and increased in frequency; at the age of 18 he was brought to the hospital. Encephalography showed porencephalia in the region of the left angular gyrus. Because of the severity of the convulsions, the region of the porencephalia was excised rather freely; it was assumed that the patient's handedness had shifted to the right hemisphere. To my great astonishment, at the end of the operation he was completely aphasic; however, he recovered his speech rather remarkably in a relatively short time.

DR. GEORGE V. N. DEARBORN: I agree with Dr. Brickner that Luciani's general conception of the configuration status of the frontal lobes is strongly confirmed by his unusual case. I have always thought that there was plenty of evidence indicating that the neopallium as a whole was fundamentally an inhibitory organ. I think that it is safe to suppose that the inhibitory action of alcohol on behavior (an effect to which these three cases bear resemblance) is due to the depressant or poisonous effect of alcohol on the nerve cells of the upper cortical layers, producing some degree of chromatolysis and disturbing the finer, cultural standards of

the mind. It is well known from the classic research begun about thirty years ago by Hodge, Austin and Sloan, Nissl, R. Hartwig and especially D. H. Dolley, that the greatest injury (chromatolysis) is done to the supposedly inhibitory parieto-frontal neopallium.

I do not altogether agree with Dr. Brickner that this patient presents any considerable amount of intellectual deterioration. The performance of the blackboard tests represents to me fairly well the work of a mind with an intelligence quotient of 100. Most men tend to limit the number of their "used" ideas and also tend to repeat several of them in the course of a series of questions like these. That does not seem to me to indicate any great degree of deterioration.

DR. LAWRENCE S. KUBIE: In his discussion, Dr. Schilder properly emphasized that the emotional and the intellectual processes are inextricably bound together. Therefore, he seems to imply that the behavior of these patients is explicable purely on the basis of an emotional change. This may be true, but one must try to understand how the emotional change came about and attempt, furthermore, to describe the change in more exact psychologic terms.

If one examines the patient closely, one is impressed by the euphoria and the mild hypomanic state which he exhibits. Such a chronic hypomanic condition means that this man must be free to an extraordinary degree from the emotions which ordinarily weigh on the spirit of normal people; he must, for instance, be free from anxiety, from any feelings of guilt or of relative inferiority and from any emotional or perhaps intellectual realization of his disabilities. Such a state of mind is encountered in two classic clinical pictures: in typical manic elation and with minor changes in certain cases of high grade feeblemindedness. But how does it happen that a puny man so elated can flex his muscles and say, "Look at my enormous strength," and can tackle without fear a husky ward attendant? Is there first a subtle intellectual change, which permits the instinctive emotional drive to express itself without any consideration of results and without the anticipation of disaster which would excite anxiety in most persons. It is obviously not enough to say that fear or guilt is "inhibited." Such a statement merely describes the end-result with a pseudoscientific word. Dr. Brickner thinks that there is some such primary reduction in intellectual perceptions. Dr. Schilder believes that the man does not demand of himself the same aims and goals which he did before his operation and that all of the changes in his behavior are emotional consequences therefore. On the other hand, Dr. Brickner speaks of the "masterly evasion" with which the patient avoids any problem which is too difficult for him. This implies that he has some dim insight into his incapacity. Furthermore, it is striking that his bland contentment is broken through chiefly when some problem is forced on him so insistently that he cannot accomplish this evasion, with the result that he is forced to face his disability. In this respect one is reminded of the tantrums of a feebleminded person, whose calm good nature can be completely disrupted when some contact with reality is sufficiently intense to break through the dimness of his perceptive powers.

The occasional emotional explosions which occurred in the cases reported by Dr. Fox seem to fit into this general pattern. In one case the short-lived attacks of anxiety apparently broke through the euphoria almost spontaneously. Perhaps this could be correlated with the extent of the operative injury.

Finally, it would be interesting to know whether Dr. Brickner has made any study of the flow of ideas in the free associations of the patient. In listening to him, one is impressed with the occasional sporadic use of words in almost a purely figurative and symbolic sense. It might be interesting, therefore, to scrutinize some of these productions much as one would scrutinize a dream.

DR. C. BURNS CRAIG: Dr. Brickner has made an important contribution on the function of the frontal lobes. A rare opportunity to make such a study was presented, and he seized it. His study has been the close-up view of an intimate friend of the patient. I should like to present the slightly different impression made on a stranger.

I have seen Mr. A. three times at intervals of six or eight months. He did not remember me and was introduced each time. We were strangers. His conduct was identical on each occasion. He "bristled" as a dog does on meeting a strange dog. His attitude was that of many wild animals and some militarists, that the best defense is offense. After a brief period of wariness he became almost pugnacious, provocatively so. He began telling how "good" he was in everything he undertook. He assumed the truculent attitude of a bully. One cannot but reflect on the fact that the village bully as a rule is a boy whose home conditions are lacking in restraint. His behavior reminded me of the juvenile boastfulness of Goliath before the children of Israel. The impression made was a combination of defense lacking emotional restraint and poor judgment in the method used to convey the impression of superiority.

To say that the censor or superego has been largely removed because of the moderately unrestrained emotional reaction is to call attention to only part of the picture. The whole personality has been seriously damaged. His complacent self-contentment and lack of insight into his condition and his lack of concern indicate markedly impaired judgment.

From my own brief and inadequate observation of this man and from Dr. Brickner's statement of the case, it seems that one might regard the frontal lobes as the areas in which terminal thinking is done and final conclusions are reached, in a word, the seat of judgment. But that was not all. The acquisition and retention of new ideas seemed faulty. This suggests that the frontal lobes play a part in the storage of memories. Furthermore, the man seemed to lack the power of concentrated attention.

DR. RICHARD M. BRICKNER: It seems to me that there is little in Dr. Woodworth's discussion for me to answer because, as I understood him, he agrees with me regarding complex syntheses. I had not considered the synthetic functions of the frontal lobe in the light of motor activity—a point of view of considerable interest.

Dr. Schilder's suggestion that the frontal lobes serve the "highest" psychic functions may be termed hierarchic. To me, these lobes have the distinction only of subserving extremely complicated functions, about which there is nothing "highest."

Dr. Schilder raises a puzzling question when he discusses the identity or the lack of it between emotional and intellectual processes. There is nothing in my view of the problem which necessarily implies a definite split between the two functions. There are, however, so many differences between emotion and intellect as they are seen in human behavior that it may be practical to consider them separately. In analyzing the case of my patient such a separation appeared to me to help to clarify all of the symptoms. I do not mean to imply a fundamental biologic separation, because one does not know whether such a division exists. However, the explanation here given applies to certain symptoms which are hard to explain on a "purely emotional" basis, if I may use such a term. In such matters as impairment of recent memory, in contrast to remote, for example, it is difficult to be certain whether or not emotional factors are of prime importance.

Dr. Schilder's use of the conception of the "finishing part of thinking" is extremely interesting. Possibly it may be alined with the synthetic theory that has been presented, though its significance may rather be in terms of the "closure" of the *Gestalt* psychology.

Dr. Oberndorf spoke of the frontal lobes as perhaps being the location of the function of inhibition. For my own part, I avoided this term scrupulously. Probably, though, in whatever sense these lobes are inhibitory, they are no more so than the rest of the cortex.

Dr. Dearborn thought that no definite disintegration is shown in my patient, but that the behavior is what he would expect of a man with an intelligence quotient of 100. I am not prepared to say that he should not expect that, but I feel certain from what I know of this man that he had a much higher intelligence quotient and was much more competent before the operation. I think that Dr. Dearborn would agree had he had the opportunity to study the patient intensively.

Dr. Craig's comments are of interest and add to the completeness of the presentation.

Dr. Kubie states my own feelings accurately in being confused as to whether the emotional or the intellectual defects come first and also in several other respects.

DR. JAMES C. FOX, JR.: I wish to point out that we thought that our first patient did not show any significant alterations of personality except for a little increase in facetiousness. As for the second patient, I want to emphasize again that the presence of a speech disorder and a certain impairment of the intelligence, the emotional reaction, which he showed might have followed as a natural consequence of the operation. One must constantly be on guard and interpret such a reaction as overfacetiousness and the type of behavior which was displayed by Dr. Brickner's patient as the individual's attempt to adjust himself to his environment; one must not identify the emotional reaction with a specific part of the brain. I think that the fight that Hughlings Jackson made throughout his entire career should be recalled; he continually pointed out that there are two effects from every local negative lesion of the brain. One is the definite negative effect—the "outfall" of function. The other, which is far more important, and which beclouds the whole issue, is the positive effect—what the rest of the healthy brain does in acting in a different manner to compensate for the loss. He speaks of this as "physiology in difficulties." This certainly applies when part of the human brain is destroyed as in lobectomy. One must be careful in trying to ascribe any particular symptom to the lack of a part of the brain which is gone.

Book Reviews

Experimentelle und klinische Studien zur Physiologie und Pathologie der Pupillenbewegungen mit besonderer Berücksichtigung der Schizophrenie. By O. Löwenstein and A. Westphal. Price, 18 marks. Pp. 181, with appendix and 101 illustrations. Berlin: S. Karger, 1933.

In this experimental and clinical study of the physiology and pathology of the pupillary movements the authors consider in great detail various factors of interest not only to the ophthalmologist but also to the neurologist. Their methods of studying these pupillary reflexes are outlined in great detail. There are three general subheadings: (1) a study of the normal physiology of the pupil and its normal psychologically controlled reactions; (2) a study of the pathology of the pupillary movements on a basis of psychopathology, and accompanying studies, and the control of the pupillary movements, and (3) the results and conclusions from both the previous subheadings.

Section 1 is subdivided into various allied factors. Under the study of the light reaction, the authors consider the time, the source, the maturity and the duration of the light reactions. They form or classify various types of these as to maximum contraction, minimum contraction and the speed of contraction, changes which appear under fatigue, the consensual reaction and changes which occur in the form of the contracted pupil. This is a consideration of normals. Continuing the consideration of normals and the effect which psychologic stimuli have on pupillary movements, the authors discuss the effect which pain, fear and the anxiety states have on them. This study of normals includes a consideration of the very fine pupillary movements which occur especially at the pupillary margin and not over the entire surface of the iris.

Section 2 is a study of the pathologic pupillary movements, especially of the movements controlled and impelled by accompanying psychopathic states and conditions. In this study consideration is given to the relationship to schizophrenia; the manic-depressive states; extrapyramidal conditions, such as postencephalitic conditions; chorea; Little's disease; generalized athetosis; progressive muscular dystrophy, Huntington's chorea; epilepsy, and other conditions, such as myasthenia gravis. The differences in the pupillary apertures on the two sides occasionally seen in normal health are also discussed.

The conclusion abstracts most satisfactorily the text which has gone before in a logical group of connected paragraphs. Some of the subjects treated, repeated in part for emphasis, are: the latent period of the pupillary reflex, the form of the reflex, the effect that fatigue has on it, the consensual reaction, the shape of the contracted pupil, the effect of psychologic reflexes on the pupillary reflexes and others.

The book is well illustrated with a large number of charts and some photographs. It is fairly well printed, and the type is legible. The binding is poor. The reviewer's copy, after the leaves had been cut, was kept together only with difficulty. The book is a worth-while experimental and clinical contribution to the study of the pupillary reflexes, of certain most important relationships in health and disease and of the factors which modify, control and are manifested by changes in the pupil and in the motility of the iris. The great experience of the authors, as presented, fills a want long felt in ophthalmic literature.

Klinische und vererbungsmedizinische Untersuchungen über Oligophrenie in einer nordschwedischen Bauernpopulation. By Torsten Sjögren. Price not stated. Pp. 121, with numerous charts and tables. Copenhagen: Levín & Munksgaard, 1932.

This monograph might well serve as a model of what a modern heredobiologic study in psychiatry should be. It is gratifying to see that Lundborg's famous

institute is continuing its important researches in this field. The need for such expert investigation of problems of heredity is the more pressing today since Rüdin's division of the German Research Institute for Psychiatry has forsaken science to indulge in pseudoscientific "race propaganda" (T. Lang: *Nationalsozialistische Monatshefte* **24**:119, 1932. Luxenburger, H.: Angewandte Erblichkeitslehre, Sozialbiologie und Rasse, *Fortschr. d. Neurol., Psychiat.* **5**:392, 1933).

This study covers an investigation of forty families. Fifty-two cases of feeblemindedness occurred in thirty-four of the families. The feeblemindedness was congenital and stationary. It was usually noticed first in the second or third year of life. The mentality of the persons affected did not progress beyond an age level of between 3 and 6 years. The subjects had not learned to read, write or calculate. Their language was dysarthric and frequently agrammatical. They had a slow and lumbering gait.

In five cases "brain punctures" were made to permit examination of brain tissue. Study of the samples so procured revealed no trace of cyto-architectonic disorder; further histologic observations were not made.

The author concludes that the hereditary transmission of this condition is probably of a recessive or monohybrid type. The anlage of the disorder seems to be transmitted more easily through the mother than through the father; at least it is more easily manifested when transmitted through the maternal side.

The monograph deals with the subject in a thorough manner. It contains a number of excellent tables and charts and a complete bibliography.

Crime and Criminals. By William A. White. Price, $2.50. Pp. viii + 276. New York: Farrar & Rinehart, 1933.

In view of the author's major contributions to the practice of and literature on criminologic psychiatry, much would be expected of the present work. While written in a lucid manner, it deals sketchily with a number of criminologic topics and is presumably for the layman rather than for the criminologist. The earlier chapters define the author's attitude toward man's position in society and make plain certain facts not yet apparent to the layman but entirely absorbed by the expert, facts which treat largely of the freudian point of view of mental mechanisms; for example, conflict, individuation, regression and the relation of crime to love, hate and guilt. In a chapter entitled "Insanity and Crime" there is a brief and lucid discussion of the problem of responsibility, which can be recommended to an unenlightened judiciary. This chapter is followed by a discussion of "good" and "bad," which is competent as a discussion but rather too philosophic for the lay reader. On the other hand, the discussion of punishment, to which two chapters are devoted, and a prognostication of future tendencies may be considered as representative of the ideas expressed by the majority of criminologists with psychiatric training. The possible exceptions lie in the case of the court or prison psychiatrist who finds that many of the ideas derived from the study of pathologic cases do not find a counterpart in the offender who is not suffering from a neurosis or a psychosis. It is to be hoped that White will some time write an entire volume devoted to the reminiscences of his court experiences, of which he gives a sample in the last chapter. The attitude of the layman toward this book is indicated by the fact that it was chosen by the Book of the Month Club. It certainly cannot harm the casual reader and may enlighten him. Its value for the psychiatrist is doubtful.

Beiträge zur Kenntnis der Narkolepsie. By Rudolf Thiele and Hermann Bernhardt. Price, 18 marks. Pp. 187, with 2 illustrations. Berlin: S. Karger, 1933.

This monograph on narcolepsy is thorough and based on a considerable number of cases. Whereas in 1924 Redlich could collect only 35 cases—11 of his own and 24 reported in the literature—by 1931 the number of cases which had been published had increased to about 150. Thiele and Bernhardt ascribe this

continuously growing number of reports not only to the increased interest of physicians in disorders pertaining to sleep but also to an increase in the incidence of the malady. Their own material consists of 31 cases seen in the neurologic clinic of the Charité in Berlin from 1925 to 1933.

In agreement with Lhermitte and Kinnier Wilson, Thiele and Bernhardt believe that narcolepsy is merely a clinical syndrome and not a disease entity. They make a distinction in their own material between "genuine" cases of narcolepsy, postencephalitic cases and posttraumatic cases. Thirty-one histories are given in full. A few others are included that do not concern narcolepsy strictly but that have some symptomatologic relationship to this syndrome. The discussion of narcolepsy is clear and critical, but it does not (except in minor details) add much to the data furnished by some of the excellent previous publications on the subject.

The Intelligence of the Prospective Immigrant. Public Health Bulletin, No. 206. By J. D. Reichard, Surgeon, United States Public Health Service. Price, 5 cents. Washington, D. C.: United States Government Printing Office, 1933.

This bulletin presents the results of a study of mental ability measured by "language" and "non-language" tests of approximately 500 persons applying for American immigration visas at Warsaw, Poland. In general the results are presented in the form of two schedules, i. e., nonlanguage and language. The results of applying performance tests alone compare favorably with those in American school children over 10 years of age. Language tests, however, gave results below those for American school children over 10 years of age, the results being strikingly lower in some language tests. Sex, schooling and age were significant factors influencing ability, whereas only slight differences were associated with race. Nonlanguage tests showed the greatest differences in association with sex, and language tests with schooling. The most consistent variation in results was associated with age, the older age groups uniformly making the poorest showing. The study is of value to all agencies, both institutional and communal, that deal with the problems of mental health associated with European immigration.

Soziologie der Neurosen; Die nervösen Störungen in ihren Beziehungen zum Gemeinschafts- und Kulturleben. By Karl Birnbaum. Price, 4.60 marks. Pp. 87. Berlin: Julius Springer, 1933.

This pamphlet represents a paper that appeared in the *Archiv für Psychiatrie und Nervenkrankheiten* (**99**:339, 1933). It contains a highly theoretical discussion of the rôle played by social factors in the neuroses. Most of the literature on the social aspects of the neuroses is based largely on general impressions. Statistical data on the subject are almost nonexistent. Birnbaum points out this fact, and his discussion forms no exception to the rule. The views he propounds add nothing to the statements to be found in the psychopathologic (and especially the psychoanalytic) literature. He has evidently made no attempt to acquaint himself with modern scientific sociology, and instead of dealing with concrete social realities he restricts himself to somewhat vague generalizations. Only German literature and German institutions—the latter largely swept away by recent events in Germany—are considered.

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